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This is to certify that the Master's thesis of

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To Mary, my wife, thank you for your love, friendship, and advice (*carpe diem*) you helped me enjoy this experience.

To our children Jacqueline and John for your love, understanding, and sacrifices, may you enjoy the fruits of this labor.

To my parents for your love, support and encouragement may you continue to take pride in your children's accomplishments.

"The masticatory system, like all biological systems, is constantly adapting to change.... Adaptability varies from patient to patient and changes from time to time in a given patient."

Michael W. Parker, D.M.D.
A dynamic model of etiology in
temporomandibular disorders.
Journal of the American Dental Association
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A LONGITUDINAL EVALUATION OF THE EFFECTS OF ORTHODONTIC
TREATMENT ON CLINICAL SIGNS AND SYMPTOMS OF
TEMPOROMANDIBULAR DISORDERS

by

John George Kharouf

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INTRODUCTION

Definition of Temporomandibular Disorders

Temporomandibular disorders (TMD) is the term officially adopted by the American Dental Association (Griffiths, 1983) to denote a group of clinical problems involving the temporomandibular joints, the masticatory musculature or both (McNeill, 1990, 1990a).

In the past, these problems have been viewed as one encompassing syndrome, however, current research suggests that temporomandibular disorders are related, distinct disorders in the masticatory system with many common features (Bell, 1982, 1990; Griffiths, 1983a).

Joint sounds are the most common symptoms found in the adult populations studied epidemiologically, and masticatory muscle pain from palpation is the most common sign (Helkimo, 1979; Schiffman and Friction, 1988). The most common initial symptom for patients seeking treatment is pain in the temporomandibular joints and muscles of mastication (Helkimo, 1974a, 1979; Schiffman and Friction, 1988). Pain is usually worsened or brought on by jaw function (Dworkin et al., 1990a). The other common symptoms are limited jaw

movement and headaches (Laskin, 1969; Bell, 1982, 1990; Solberg, 1982; Clark, 1987; McNeill, 1990, 1990a, 1991).

Purpose of the Study

The purpose of this study is to assess the effects of orthodontic treatment on the clinical signs and symptoms of TMD.

The objectives are to determine which components of the Helkimo Indices change with orthodontic treatment and to determine if these changes are exacerbated or alleviated by orthodontic treatment.

Statement of the Problem

Ever since Costen (1934) first brought to the attention of the dental and medical professions that displaced condyles could cause orofacial pain, dentists have been treating and searching for the etiology of TMD. Many theories and therapies for TMD have been proposed through the years. At present, the literature shows that the exact etiology remains unknown. In addition, the explanations for various successful treatment modalities demonstrate that the reasons for successes are not well understood.

In the 1950's, orthodontics began to be implicated as a cause of TMD (Thompson, 1956). Since that time, the "opinion" that orthodontic therapy was a possible "preventive" TMD treatment has been countered with the

opinion that orthodontics is a cause of TMD (Greene, 1982, 1988; Barbat, 1992). Much of this opinion was based solely on "clinical impressions" (Reynders, 1990; Behrents and White, 1992).

However, as TMD therapy evolved in the late 1960's through the 1980's, it became evident that treatment of the occlusion as a sole cure for TMD was unsupported by research (Laskin, 1969; Goodman et al., 1976; Clark, 1985; Seligman et al., 1991).

In addition, the complexity of the disorders have become clearer with the advent of computed tomography (CT) and magnetic resonance imaging (MRI).

With time, the claims that various orthodontic treatment appliances and therapies are etiologic factors in the development of TMD have increased in number (Reynders, 1990). The consequence of these "case studies" and "clinical viewpoints" was a well known law suit (Brimm vs Dr. X). Today, many general dentists and orthodontists continue to believe that malocclusion and various orthodontic treatment practices predispose patients to developing TMD (Greene, 1988; Just et al., 1991).

An initial effort by Sadowsky and others (1980;1984) began to investigate not only the etiology of TMD, but more specifically, the relationship between TMD and orthodontics (Sadowsky, 1992). Other research which has since followed

continues to disclaim orthodontics as a major etiologic factor of TMD (Sadowsky, BeGole and Polson, 1980; 1984; Larsson and Ronnerman, 1981; Dibbets and van der Weele, 1987, 1991; Dahl et al., 1988; Kremenak et al., 1992, 1992a). Unfortunately, little longitudinal data is available to aid in establishing the risks and benefits of orthodontic treatment.

The longitudinal TMD study started in 1983 at the University of Iowa has resulted in evidence suggesting that orthodontic treatment is not a major etiologic factor of TMD (Kremenak et al., 1992, 1992a). What then are the possible TMD etiologic factors in young adult orthodontic patients?

Longitudinal data were collected on adolescent and young adult orthodontic patients enrolled in the Iowa TMD Study, a majority of whom were unaffected with regard to signs and symptoms of TMD during the prescribed orthodontic therapy. However, a small number of these patients had an improvement in the measured clinical signs and symptoms of TMD by the end of treatment, and an even smaller number had a deterioration of the evaluated areas with orthodontic treatment. A descriptive analysis of these three major groups of patients suggest pretreatment patient attributes which affect the measured pretreatment signs and symptoms of TMD.

Goals of the Study

Hypothesis

The hypothesis tested in this study was that all orthodontic patients remain unchanged for the anamnestic index, and five components of the clinical dysfunction index of the Helkimo Indices from pretreatment to posttreatment.

Specific Goals

1. Evaluate the inter-examiner reliability between two examiners at the beginning of the study and one year later.
2. Describe the subjective symptoms from the anamnestic portion of the Helkimo Indices.
3. Evaluate the clinical dysfunction portion of the Helkimo score by dividing the clinical dysfunction index into its five components:
 - A. impaired range of movement/mobility index
 - B. impaired TMJ function
 - C. muscle pain
 - D. TMJ pain
 - E. pain on movement of the mandible
4. Determine where changes occur within the Helkimo Clinical Dysfunction Index score for all patients for whom a pretreatment, during treatment, and posttreatment score had been collected, including those patients whose clinical dysfunction signs improved and

those whose signs worsened during the course of orthodontic treatment.

5. Determine the influences of the following pretreatment patient attributes on the components of the clinical dysfunction index:

1. Angle Classification
2. Overbite
3. Overjet
4. Crossbites

REVIEW OF THE LITERATURE

Epidemiology

Definition of Epidemiologic Signs and Symptoms

The purpose of reviewing the epidemiologic literature on TMD is to determine the prevalence of the signs and symptoms of TMD in the nonpatient population. This will provide a better understanding of the overall prevalence of TMD and its clinical significance to orthodontics.

One problem which has hindered good research is the lack of a precise definition of temporomandibular disorders along etiological (cause) or morphological (affected organ) lines (Greene, 1982; Mohl, 1991). Because of the complex nature of this disorder, a simple cause-and-effect paradigm is insufficient. A dynamic model of possible etiologies specifically for myogenous and certain arthrogenous TMD has been proposed which includes factors that affect a patient's adaptability in balance with factors which predispose a patient to hyperfunction (Parker, 1990). This model and similar models have attempted to account for the interaction of the individual and the complex, multifactorial etiology of TMD (Moyers, 1985; Vandas, 1988). As a result of this

lack of a definition, temporomandibular disorders have been described on the basis of signs and symptoms.

The characteristic symptoms of temporomandibular disorders most commonly included in the various descriptions are:

1. functional jaw pain, either TMJ or muscle,
2. temporomandibular joint incoordination,
3. restriction of range of mandibular movement,
4. temporomandibular joint sounds, and
5. recurrent headache.

(Solberg et al., 1979; Green, 1982; Rugh and Solberg, 1985; Clark and Solberg, 1987)

Once these signs and symptoms were established, evaluation could be based on some common ground for defining temporomandibular disorders. This understanding eventually led to the Helkimo Indices (Helkimo, 1972) and other epidemiologic indices for TMD (Greene, 1982a; Rieder and Martinoff, 1983a; Friction and Schiffman, 1986, 1987; Pullinger and Monteiro, 1988). These indices have aided in the objective assessment of the prevalence of TMD.

The Helkimo Indices

Description of the Helkimo Indices

The Helkimo Indices were developed by Marti Helkimo in Sweden in 1972 for the epidemiologic assessment of TMD signs and symptoms in the Lapp population of northern Finland

(Helkimo, 1974a). Helkimo established a weighted, systematic method for classification of the disturbances and determination of the prevalence and the severity of clinical dysfunction. The Helkimo Indices consist of an anamnestic, or self-appraisal, portion of 10 questions, a clinical dysfunction examination, and an occlusal dysfunction evaluation based on dental casts. The Iowa TMD study has, since its inception, utilized only the anamnestic and clinical dysfunction portions of the indices.

The anamnestic dysfunction index is based on the patient's opinions in response to 10 closed (yes/no) questions relating in a subjective way to the five areas of the clinical dysfunction index. The anamnestic dysfunction index is scored as follows (Helkimo, 1974a). A_i0 denotes the complete absence of subjective "symptoms of dysfunction of the masticatory system" (Helkimo, 1974a, pg. 107). A_iI denotes mild symptoms including temporomandibular joint sounds such as clicking and crepitation, and a feeling of stiffness or fatigue of the jaws. A_iII denotes "severe symptoms of dysfunction" (Helkimo, 1974a, pg. 107). One or more of the following symptoms were reported in the anamnesis: "difficulty in opening the mouth wide, locking or luxations", pain on mandibular movement, facial and jaw pain (Helkimo, 1974a, pg. 107).

The clinical dysfunction index is the composite score which is derived after signs are elicited from an examination. It is based on the common clinical symptoms reported in the literature. These five components of the clinical dysfunction evaluation are: A. impaired range of movement/mobility index, B. impaired TMJ function, C. muscle pain, D. temporomandibular joint (TMJ) pain, and E. pain on movement of the mandible (Helkimo, 1972,1974a).

The index for range of movement of the mandible is based on four different mandibular movements: maximum opening plus overbite, maximum right and left laterotrusion, and maximal protrusion plus overjet (Helkimo, 1974a). Each movement is given a score of 0 (normal range of movement), 1 (small range of movement), or 5 (severely impaired range of movement). Movements are summed and given a final value of 0, 1, or 5 .

Impaired function of the TMJ is scored as follows, 0 if "a straight path of mandibular opening and closing without palpable TMJ sounds" occurs, 1 if any "palpable TMJ sound or visible lateral deviation ($\geq 2\text{mm}$) of the path of mandibular opening or closing" occurs, or 5 if there is "locking or luxation of the TMJ" during the mandibular mobility movements (Helkimo, 1974a, pg. 103).

Pain on movement of the mandible is scored as follows: 0 if "all movements are performed without pain", 1 if pain

occurs in only one movement, or 5 if "two or more movements produced pain" (Helkimo, 1974a, pg. 103).

Muscle pain is determined if a particular muscle is "clearly tender to palpation", in other words, muscles are either marked positive or negative to palpation (Helkimo, 1974a, pg. 103). The final score is determined as follows: 0 "if none of the muscles are tender to palpation", 1 if one to three areas of palpation are tender, or 5 if 4 or more muscle areas are tender to palpation (Helkimo, 1974a, pg. 106).

Temporomandibular joint pain is elicited using a similar palpation method as the muscles of mastication. The score is determined as follows: 0 for temporomandibular joints not tender to palpation, 1 for temporomandibular joints uni- or bilaterally tender to lateral palpation, and 5 for uni- or bilateral tenderness to palpation from the external meatus, that is, posterior palpation pain (Helkimo, 1972, 1974a).

Each of the five clinical components weighted score of 0, 1, or 5 is then summed giving a total dysfunction score between 0 and 25. This score is used to derive an overall level of clinical temporomandibular dysfunction. There are four levels of clinical dysfunction described as follows. "0 points = Dysfunction group 0", D_10 , which is the absence of clinical symptoms, "1 to 4 points = Dysfunction group 1",

D_iI, which includes only mild symptoms of dysfunction, "5 to 9 points = Dysfunction group 2", D_iII, which includes at least one severe symptom combined with 0 to 4 mild symptoms or five mild symptoms alone for a moderate dysfunction level (Helkimo, 1974a, pg. 106). The severe dysfunction level begins with "10 to 13 points = Dysfunction group 3", D_iIII. This score includes "2 severe symptoms combined with" any of the mild symptoms for an overall severe dysfunction level (Helkimo, 1974a, pg. 106). "15 to 17 points = Dysfunction group 4", D_iIII, is a severe dysfunction level which includes three severe symptoms, and "20 to 25 points = Dysfunction group 5", D_iIII, is a severe dysfunction level of four or more severe symptoms (Helkimo, 1974a, pg. 106).

The major advantage to the Helkimo Indices is that they are a simple, yet powerful epidemiologic indices which assign a numeric value to the severity of the dysfunction which can then be more readily evaluated and statistically analyzed for individuals and samples. This is not to say that a "score of 5 means the condition is 5 times more severe than the score one" (Helkimo, 1974a). They are nominal scale indices designed to prevent sums of moderate scores from exceeding the score of a single severe sign. In this way, the indices safeguard against erroneous registration or overestimation of a sign (Helkimo, 1974a). There is limited ability for examiners who misinterpret the

specific signs measured to completely bias the results due to the summation of various observations into an index score. Muscle or TMJ palpation are most likely to be influenced by the examiner which could effect the final dysfunction score of an individual. Another advantage is that the Helkimo Indices are divided into subjective symptoms and objective signs, by a questionnaire and clinical exam, which therefore facilitates comparison of studies because of its use of fixed and graded clinical signs and symptoms.

The Helkimo Indices have discriminating power as indices because severe TMD patients are clearly identified. In a comparison of the anamnestic and clinical dysfunction indices, 75% of the subjects who felt they had severe symptoms had at least one severe clinical sign, and 44% had two or more severe signs (Helkimo, 1974c). Only 21% of the subjects without subjective symptoms were found to have severe signs. By Helkimo's conclusion then, the Helkimo Indices are valid as descriptive epidemiologic indices (Helkimo, 1974b).

However, in a similarly designed study of 222 dental and dental hygiene students, only 21% of the subjects with pain upon clinical examination in the TMJ, muscles, or head realized it (Pullinger et al., 1988a). Increased frequency of agreement between recognition of symptoms and the

clinical presence of signs was evidenced by 50% of the subjects with four or more painful muscle sites.

Difficulties in Assessment of Temporomandibular Disorders

The multifactorial etiology of TMD and lack of clear defining characteristics have made the development of a comprehensive method of assessment a complex problem (Rinchuse and Rinchuse, 1983). Standardizing the method of patient evaluation and scoring is an important link in establishing the reliability and validity of observed clinical signs. This is especially important when making comparisons between two or more studies using similar methodologies but with different observers (Solberg, 1982). Comparing the severity of symptoms from one study to another will always be less than ideal because of the subjective nature of the ratings. However, it is subjective symptoms which cause patients to seek treatment. Problems of subjectivity in the evaluation of clinical signs are very difficult to control, and will always be present to a degree in clinical studies (Gross and Gale, 1983).

The importance of realizing that these results do not necessarily identify people who are at risk of developing TMD can not be overstated (Greene, 1982). We simply do not know the natural course of TMD; it has not been documented for the non-patient population. Research dealing with the

course and duration of TMD is limited. In 1981, Rasmussen reported on a six-year longitudinal study documenting the course and duration of the disorder on 119 patients with temporomandibular arthropathy, some of whom had semiopaque arthrography of the TMJ. Patients went through three stages each with two phases based on the progress of subjective symptoms (Rasmussen, 1980, 1981a, 1981b). The stages were initially clicking and locking, then TMJ pain and restriction, followed by nonpainful residual symptoms, and finally complete disappearance of symptoms. In general, the stages had an average duration of 4 years, 1 year, and 6 months respectively. The problem with this type of research is that the signs and symptoms may be transient and self limiting, or resolving without serious long-term effects.

Most temporomandibular disorders appear to be mild and self limiting; however a number of patients develop a chronic pain condition. Little is known about which signs or symptoms will progress to more serious conditions, or the significance of each of these signs and symptoms. Predisposing factors have not, as yet, been conclusively determined to accurately identify which patients are at risk of developing temporomandibular disorders, nor is it clear which factors are etiologic or contributory in nature (Zarb, 1985). Data are not yet available to help the clinician establish who is in need of treatment and who might be best

left alone. Although a large percentage of the population have signs and symptoms, it is estimated that only 4 to 7% are so severe as to require treatment (Solberg et al., 1979; Locker and Slade, 1988; Dworkin et al., 1990).

Therefore, the following review of epidemiologic studies merely point out many common features of this chronic disorder, without pinpointing a predominant etiologic factor of TMD in the population as a whole.

There are also problems of reliability especially of anamnestic questionnaires and problems with social and psychologic differences between men and women.

In addition, many studies do not qualify the joint sounds. Clearly, clicking and crepitus have different clinical implications and levels of severity. Helkimo claims reliability of the clinical dysfunction index is "relatively good" (Helkimo, 1974a).

Temporomandibular disorders, as we presently understand them, are largely dependent on the adaptability of the patient (Roth, 1976; Rugh, 1991). Therefore, many subclinical effects may be present before they manifest themselves to where they can be detected (Helkimo, 1974b). In essence, to come up with a certain conclusion regarding the diagnosis of TMD, we would need to know the patient's future adaptability. Because this can never be known,

clinicians must accept the limitations of these types of TMD indices and studies.

Clinical research in the treatment of TMD is difficult in many respects because of the impossibility of treating and not treating the same patient to evaluate treatment results. In this study, no effort was made to follow an untreated control group longitudinally due to the extreme difficulty in this endeavor as well as medicolegal ramifications. Alternatively, from the literature review of untreated populations and orthodontically treated samples, the incidence and frequency of the signs and symptoms and the natural course of these signs and symptoms over time can be generalized. This previous data base will be used for discussion of this study's results and their implications.

Criticism of the Helkimo Indices

The Helkimo Indices were an important stepping stone in the development of an objective method for TMD assessment. They do however have some shortcomings (Carlsson et al., 1980; Greene and Marbach, 1982; Van der Weele and Dibbets, 1987). For example, the patient is required to recognize certain characteristics which the researcher has to then interpret. Pain is an example of this problem. One can ask about it, attempt to palpate it, but the researcher can not develop an accurate assessment of its effect on the person's

life. The index only notes the presence or absence of pain, not the intensity or duration (Helkimo, 1974a).

Additionally, there are inherent problems in the level of detail or sophistication used to measure various signs and symptoms. For example, without the use of a pressure algometer, muscle palpation can not be standardized which increases examiner error in recording it (Jaeger and Reeves, 1986). Likewise, TMJ sounds can be listened to with a stethoscope for a greater level of investigation although at least one investigation found it less accurate than palpation (Dworkin et al., 1990). Neither of these considerations have been implemented into this study. As a consequence of this level of subjective evaluation by the examiner, a certain level of errored responses are inherently involved in the results.

Such evaluation difficulties have made some researchers question the Helkimo's "mild dysfunction" rating and its clinical significance (van der Weele and Dibbets, 1987; Schiffman et al., 1990). Any one sign found, places the subject into this initial level of clinical dysfunction. Defining any one sign as a "mild dysfunction" may not be clinically accurate. It is not a disorder by definition if only one subclinical sign is present. In addition, no attempt has been made to differentiate what particular type of TMD, if any, the indices focus on. This, in light of the

multiple, defined temporomandibular disorders, may help explain why some of the signs and symptoms are inconclusive for diagnosis of "TMD".

The Helkimo Indices have only been validated as descriptive epidemiologic indices, that is, they determine the occurrence of disease (Helkimo, 1974a). However, Helkimo did select the most common or classic signs and symptoms of TMD based on "informed clinical judgement" (Helkimo, 1974a). They are pain in the muscles of mastication and TMJ's, decreased range of motion of mandibular movement, and TMJ sounds. This triad of signs, while not completely diagnostic in themselves for TMJ, continue to have importance in the diagnosis of TMD.

The Iowa TMD study is also incomplete with regard to the original Helkimo exam. The occlusal dysfunction index portion was never included in the study. The occlusal dysfunction index evaluated and weighted the number of occluding teeth, lateral interferences, and the effect of a centric relation/centric occlusion shift. Recent investigations as well as Helkimo's original epidemiologic work on the Lapp population show that the CR/CO slide may be a potentially important factor for the etiology of TMD in young patients (Helkimo, 1974b; Solberg et al., 1979; Ingervall et al., 1980; Egermark-Erikson, 1983, 1987; Magnusson et al., 1991; Seligman and Pullinger, 1991).

However, most research evaluating occlusal factors as etiologic factors of TMD show they have a minor role (Sadowsky and BeGole, 1980; Seligman et al., 1988; Smith and Freer, 1989).

Epidemiologic Investigations of TMD in Untreated Populations

Prevalence in Children and Adolescents

In one of the original surveys of TMD and its importance to orthodontics, 304 preorthodontic patients 6 to 16 years of age, were screened for clicking and muscle tenderness to palpation, Angle classification, and open or closed bite (Williamson, 1977). Clicking, muscle tenderness or both, unilaterally or bilaterally was found in 35.2% of the cases. The most common sites of muscle involvement were the lateral pterygoids (54.2%), medial pterygoids (30.8%), and the masseter (25.2%). In addition, 54% of the symptomatic patients had an overbite of 50% or more.

In a similar type of exam involving 200 consecutive preorthodontic treatment patients, the prevalence of clinical dysfunction increased from 40% in children younger than 10 years old, to 76% in patients over 17 years old (Owen, 1977).

A baseline of TMD symptoms was presented for a longitudinal study of 1018 12-year-olds from South Wales who were preselected on the basis of criteria of occlusal

conditions of interest to orthodontists (Mohlin et al., 1991). Signs of TMD were found in 46% of this group of children all of whom had some sort of malocclusion. The results indicate that less than 8% had mildly impaired mandibular mobility, 20.4% had some impaired TMJ function (including 7.1% with TMJ pain upon palpation and 2.7% with joint sounds), 26.6% of the children showed tenderness with masticatory muscle palpation (again it was the lateral pterygoid with the highest frequency), and pain on movement was found in 12%.

In a cross-sectional study of the relationship between malocclusion and the signs and symptoms of TMD, Egermark-Eriksson and others found nearly as many subjective complaints in 7, 11, and 15 year old Swedish children (total number 402) as those found in the adults studied (Egermark-Eriksson et al., 1981, 1983). Of the total group, 16 to 25% had occasional, mild subjective symptoms which increased in prevalence with age. Again the most common subjective symptoms were occasional joint sounds (7 to 20%) and occasional pain or fatigue in the jaws or face (29 to 59%).

Clinical signs of dysfunction were found in 30% of the youngest group and 60% of the oldest group. Joint sounds (10 to 19%) and muscle tenderness (20 to 43%) were the most common signs.

The bias of course is that most of the signs and symptoms were occasional and mild, which may explain why children are usually not thought of as having TMD. In addition, the increased prevalence of clinical symptoms with age was mainly due to the increases in joint sounds and muscle tenderness. Only 1% reported that the symptoms were present on a frequent basis. Like Helkimo, they found good correlation between subjective and objective symptoms.

Applying the Helkimo clinical dysfunction index, children displayed severe dysfunction only occasionally. On the other hand, mild to moderate dysfunction was noted in 33% of the 7-year-olds and 66% of the 15-year-olds. Headache appeared as frequently in children as in adults. Correlations between parafunctional habits and the dysfunction index indicated that bruxing individuals were at greater risk for development of symptoms.

In a follow-up longitudinal study, 119 of the two youngest groups came to a new examination 4 years later, and 119 of the oldest group, 15 years old initially, were examined 5 years later (Egermark-Eriksson et al., 1987, 1990). Of the 20 year olds, 15% had received orthodontic treatment. Those who had received the corrective orthodontic treatment showed no higher prevalences of occlusal interferences, subjective or clinical signs of TMD than the untreated subjects. Of this oldest group, only

5.0% had Helkimo D_i of II or III at both examinations. As in the earlier study, no strong correlations, above 0.3, were found between morphological malocclusions and the presence of frequent headaches, bruxism, symptoms or clinical signs of TMD (Egermark-Eriksson et al., 1987). They concluded that while no single occlusal factor can be attributed as the cause of TMD, certain aspects of malocclusions, for example posterior crossbite or anterior openbite, appear to be potential risk factors for future TMD development.

In the most recent follow-up study of this same group 10 years after the initial evaluation, 293 of the original 402 answered a questionnaire concerning orthodontic treatment and symptoms of TMD (Egermark and Thilander, 1992). Clinical exams were performed on 83 of the 25 year olds, 37% of whom had orthodontic treatment. Most of the orthodontic treatment was completed between the ages of 13 and 16.

Subjective symptoms increased in all age groups, but were more frequent in untreated subjects. In general, TMJ sounds occurred slightly more frequently in untreated subjects 10 years later, although it should be noted that the changes from clicking to not and vice versa occurred irrespective of orthodontic therapy. Orthodontically treated subjects had lower clinical dysfunction scores 10

years later with over 68% having no clinical signs of TMD, 25% being D_{I} I, and 8% having severe signs of dysfunction. This is compared to the untreated group who had a decrease in the number of subjects with no signs to less than 25%, and increases of D_{I} I to 52%, and D_{I} II-III to 23%.

In a study of 440 Swedish children age 7 to 14 years of age, Nilner and Lassing also found similar levels of subjective symptoms, with 36% reporting one or more symptoms, including 15% who noticed clicking sounds (Nilner and Lassing, 1981). Orthodontic treatment was completed in 7%, and 8% were currently under treatment.

In the clinical examination, 72% of the children had one or more of the following clinical signs: TMJ sounds, less than 40mm opening, or TMJ or muscle tenderness upon palpation. Many of the children (64%) claimed to have pain upon palpation of masticatory muscles. The posterior digastric (35%), which they stated was hard to accurately palpate, and the temporalis attachment were the most frequent areas of pain. Pain on palpation of the TMJ occurred in 39%; 31% lateral and 22% posterior. Pain occurred with mandibular opening in the joint in 6%. Opening irregularity occurred in 32% of the sample.

In a second study, Nilner found that 41% (including 28% with clicking sounds) of 309 Swedish adolescents 15 to 18 years old reported symptoms in the masticatory system ,

which is slightly higher than the 7 to 14 year old subjects. Orthodontic treatment was completed on 25% and 5% were currently undergoing treatment.

Upon clinical examination, 34% had pain from palpation of the temporomandibular joints, nearly equal posterior and lateral. Clicking, found in 14%, increased with age, but was not interpreted as a sign of ageing. Muscle tenderness to palpation occurred in 55% of the subjects, with the posterior belly of the digastric (26%), the lateral pterygoid (27%), and the attachment of the temporalis muscle (20%) being the most common sites. Irregular movements or deviation upon opening were seen in 50% of the subjects. Of the total group, 77% had one (37.9%), two (24.6%), or three (5.2%) of the following signs: TMJ sounds, decreased maximum opening (less than 40mm), and TMJ and muscle tenderness upon palpation (Nilner, 1981a). In other words, more symptoms (some more serious) appeared to develop with age.

Prevalence in Young Adults

The frequency of signs and symptoms in 253 Swedish inductees who ranged in age from 18 to 25 years were determined by both questionnaire and examination (Molin et al., 1976). Results from the anamnestic portion indicated a low frequency of symptoms in general. Clicking in the TMJ, reported by 14% of the subjects, was the most frequent finding followed by clenching of the teeth.

When the sample was divided into subjects (12%) who reported subjective symptoms of pain, limited opening or locking and those who did not have these symptoms, there was a statistically significant difference for joint sounds and muscle palpation tenderness. Likewise, when the sample was divided into subjects who had the following clinical signs (28%), pain with mandibular movements, deviations with opening and tenderness of the muscles or TMJ, and the remaining subjects regarding the occurrence of various reported and clinical symptoms, the most common differences, statistically significant, for the clinical dysfunction group were: TMJ sounds (anamnestically) and occlusal disturbances (clinically).

Another study also found in patients with clinical symptoms, balancing side interferences were the only occlusal disturbance that was significantly correlated with the symptoms of dysfunction (Molin et al., 1976).

Questionnaires and examinations on 739 UCLA college students between 19 to 25 years old (mean 22.5), found only 20.1% of the sample were free of all signs or symptoms of TMD (Solberg et al., 1979). From the questionnaire, 16.8% reported only one symptom and 9% had two or more.

Upon clinical examination, women had a 10 to 15% higher occurrence of temporomandibular joint sounds (found in 28.3% of the total overall) and muscular tenderness (found in

34.2% of the subjects overall; 30% one to three sites and 4% four or more sites) than men (Solberg et al., 1979). TMJ tenderness was found in only 5.3% of the subjects. The muscles most frequently found to be tender were the lateral pterygoid (27%), the posterior digastric (7.5%), and the superficial portion of the masseter (5.2%).

In addition, 46.3% of the men and 57.8% of the women had at least one sign of dysfunction. However, both men and women reported the same overall prevalence of symptoms (26%). Clicking and headache were most likely to be isolated symptoms, and pain on opening and chewing were most often accompanied by other symptoms. Although it could not be explained, twice as many men (26.9%) as women (13.3%) were free of dysfunctional signs and symptoms. Women appeared to have a higher prevalence of signs associated with dysfunction, yet they were not more symptomatic.

The prevalence of TMD symptoms in 285 17 year old Swedish youths living in Skelleftea were studied (Wannman and Agerberg, 1986, 1986a). Using the Helkimo anamnestic dysfunction index, the most commonly reported symptom was joint sounds (13%) and tiredness in the jaw (6%). Prevalence was 20%, that is, a report of any symptom of TMD. No statistically significant difference was found between the boys and girls. In addition, 42% of the subjects had received some sort of orthodontic treatment, while 35% had

some sort of malocclusion clinically. Defining the subjects according to Helkimo (D_i0 to D_iII), 80% had an absence of subjective symptoms, 13% had mild symptoms, and 7% had severe symptoms.

In terms of prevalence of signs of TMD, masticatory muscle tenderness to palpation was the most frequent finding (41%), followed by joint sounds (22%). Girls were statistically more often tender to palpation than the boys. Signs of TMD were found in 56% of the subjects, girls significantly more often than boys.

Over half (53%) of the boys and 35% of the girls had no clinical signs of dysfunction as defined by Helkimo. Total mild and moderate dysfunctions averaged 42% and 15% respectively, but were more common in the girls than the boys.

A study on the prevalence of temporomandibular disorders in University of Minnesota nursing students 22 to 25 years old was conducted using both the Helkimo and Craniomandibular (CMI) indices (Schiffman et al., 1990). Clinically determinable temporomandibular disorder was found in 69% of the students, however only 6% had symptoms severe enough to warrant treatment. The evaluation found the prevalence of disorders of the joint, muscle, or both to be 19%, 23%, and 27% respectively. It is interesting to note that 6.7% of the sample had received TMD treatment.

Magnusson in a study of 119 20-year-olds, tried to assess the demand for TMD therapy. Based on clinical judgement, only 3% of the subjects desired TMD therapy for their signs and symptoms although 27% were judged to be in need of treatment (Magnusson et al., 1991).

Prevalence in the General Population

In a cross-sectional epidemiologic study of the signs and symptoms of TMD, both internal derangements and muscle disorders, evaluated 250 22 to 25 year old nursing students with both a questionnaire and a clinical examination which allowed comparison of the Helkimo Indices and the Craniomandibular Index (Schiffman et al., 1990).

The anamnestic portion showed that joint sounds (44%) were the most common subjective symptom. Of the entire sample, 43% were symptom free and 34% had severe symptoms according to the anamnestic portion of the index (Schiffman et al., 1990).

Clinically, 7% were completely symptom free, 34% were D_iI, 33% were D_iII, and 26% were D_iIII. The most common clinical sign again was joint noises which were found in 45.7% of the subjects. However, by defining normal joints as those which would not make sounds, allow opening greater than 40 mm, lateral movements greater than 7mm, and silent deviations on closure, 54% had bilateral normal joints.

By using the craniomandibular index, used to objectively estimate the level of clinical mandibular dysfunction, 31% of the joints were normal and 69% of the sample had a clinical TMD: muscle (23%), joint (19%), or both (27%) involved (Schiffman et al., 1990).

In 1972, a random sample of every 35th person between 15 to 74 years of age in Umea, Sweden was made using a mailed questionnaire (Agerberg and Carlsson, 1972). The purpose was to determine the frequency of functional masticatory disorders and associated factors. The results showed that 57% of the 1,106 questioned had at least one of the symptoms of temporomandibular disorders and almost one third had two or more symptoms.

Pain with maximum opening was reported by 12%. Women reported having impaired mandibular movement (7%) and clicking or crepitation in the TMJ (39%) more often than men. In addition, women sought therapy for these disorders more often than men.

Pain with mandibular movement occurred most often in the 15 to 29 year olds (16%) and decreased in frequency with increasing age. Limitations of mandibular movements were about the same for all age groups, about 7%.

TMJ sounds were the most common symptom occurring in 39%, with no differences between age groups.

Sex and age distribution of pain and other symptoms of dysfunction were about even. Women had symptoms slightly more often than men, however the overall incidence was about the same as men. They did find though that women sought treatment more often than men, which could possibly explain why prior clinical studies showed women as having a higher incidence of TMD than men.

An epidemiological clinical study on 328 "genealogic genuine" Finnish Lapps aged 15 to 65 years old, which used the Helkimo Indices, found that 12% were clinically symptom-free, of whom 65% stated they were subjectively (anamnestically) symptom free (Helkimo, 1972, 1974,a,b,c). Of the symptomatic individuals 41% had mild clinical symptoms, 25% had moderate symptoms, and 22% had severe symptoms. Symptoms of dysfunction were equally divided among men and women, and varied slightly with age. The lowest frequencies of both anamnestic (patient recollection) and clinical symptoms were found in the youngest age group (15 to 24 years old). According to the anamnestic index, 43% of the subjects reported they were symptom free, however, only 18% of them were also clinically symptom-free. In general, the higher the number of teeth lost, the more likely it was to find symptoms.

Findings from a well designed epidemiologic study of temporomandibular disorders in a random sample of 583

Finnish workers 18 to 64 years old were similar to the earlier studies on the Lapps (Swanlijung and Rantanen, 1979). Of those surveyed, 58% were found to have at least one "subjective" symptom of dysfunction, 28% reported just one and 16% reported 2. TMJ sounds were the most common symptom and was reported by the group with the highest number of occluding natural teeth. Of these people, 5% had actually received treatment for TMD.

Clinically, 41% displayed at least one sign of dysfunction, although clinical symptoms were significantly lower in the subjects under 35 years old. Again, few differences were present between males and females .

A 1983 cross sectional study of 1000 American private practice patients who ranged in age from 3 to 89, mean of 40.5 years old, reported the prevalence of common signs of mandibular dysfunction (Gross and Gale, 1983). Their results for patients between 10 and 79 years old indicated 6.2% had less than 37 mm interincisal opening, 18.7% had muscle/joint palpation pain, and 36.2% had joint sounds. Interestingly, only 0.6% had all three signs however they were not in pain, 1.3% had joint sounds and limited opening, and 6.8% had joint sounds and muscle/joint pain. Of the entire sample, 34.7% had joint sounds, "palpable irregularities", which was the most common sign.

In a study of prevalence of signs and symptoms of TMD in 1,040 private practice patients aged 13 to 86 from Newport Beach, CA, anamnestic results indicated more women than men felt they had difficulty in opening their mouth widely (17.9% verses 7.6%) and that this affected those less than 30 years old more often (21.7% versus 12% for the remaining older age groups) (Reider et al., 1983, 1983a). Subjective clicking or popping joint sounds, as in previous studies, was the most frequent symptom. It decreased in frequency with age from 44.5% in those less than 30 years old to 22.6% in those over 60 years of age. Crepitus on the other hand increased with age from 13.9% in those less than 30 years old to 16.9% in the over 60. Subjective muscle fatigue was reported by 20.7% of the less than 30 patients and decreased to 8.4% in the over 60 year olds. Every one of the questions elicited a greater frequency of response from females than from males.

Clinical signs were recorded but not ranked according to severity, and muscle and occlusal signs were recorded only if there was "extensive involvement". Of the entire sample, 47.4% had joint noises. Women had statistically significant higher frequencies of: deviated opening, joint sounds, joint pain, and muscle pain. Occlusal signs had low prevalences ranging from 2 to 18% with no significant differences between men or women.

In a similar study to Mohlin's (1976) on Swedish men with a median age of 32 years, 60% of the subjects had one or more clinical symptoms of dysfunction. Positive correlations were found between the symptoms and balancing contacts and interferences in the retruded mandibular position (Ingervall, et al. 1980).

A random sample of 677 Canadian adults aged 18 to 65 and over living in Toronto used a telephone survey questionnaire to estimate the prevalence and distribution of symptoms associated with TMD (Locker and Slade, 1988). Overall, 48.4% of the sample reported one or more of the nine symptoms in question. The most frequent symptom was joint sounds (25.4%) followed by stiffness or fatigue of muscles upon awaking (21.4%) and an uncomfortable bite (20.8%). TMJ pain either with function or at rest was found in 12.9 % of the subjects. In addition, subjects who reported pain were more likely to have other symptoms than those not having pain in the TMJ's. Based on Helkimo's anamnestic index, 58.1% of the subjects had no symptoms, 23.1% had one or more mild symptoms, and 18.6% had severe symptoms of dysfunction. Statistically significant differences between men and women were found in only three of the nine symptoms investigated, pain in and around the ears, joint sounds, and tenderness of masticatory muscles on

waking. In all three, women responded positively more often.

In the survey of potential risk factors, significant associations were found between subjects having one or more of the symptoms investigated and grinding at night, habitual clenching during the day, and frequent stress (Locker and Slade, 1988). History of trauma and orthodontic treatment were not found to be associated with symptoms.

When compared to the study of Swanljung and Rantanen (1979), 50.4% of the Canadians reported symptoms compared to the 58% of the Finnish. Regarding the relationship between the prevalence of symptoms and age, persons under 44 years old had a higher prevalence of symptoms than those 45 and over.

In a study of 222 freshman dental and dental hygiene students of mean age 23.9 ± 3.2 years (range 19 to 40) assessed for TMD by questionnaire and clinical examination, only 14% reported masticatory muscle pain and 35% reported TMD symptoms, although 75% of these were mild in nature (Pullinger et al., 1988a).

Clinically, according to the Helkimo clinical dysfunction index, 41% were symptom free, 41% had mild impairment, 17% had moderate and 1% had severe impairment.

In a controlled epidemiologic study of TMD in the United States, 1,016 subjects aged 18 to 75 were evaluated

for signs and symptoms of TMD by both questionnaire and clinical exam (Dworkin et al., 1990). The study evaluated three groups of patients: those who were symptomatic and seeking treatment, randomly selected patients who reported TMD pain, and persons within the community who are free of pain. They found 12.1% of the random community sample reported TMD pain, that is, pain in the jaw muscles, joint in front of the ear, or inside the ear within the past 6 months. The clinical exam was performed by four dental hygienists who were rigorously calibrated and who had "excellent" reliability. There were no significant differences found by age (mean was 39 years), ethnicity, or living arrangements in the clinical findings.

There were statistically significant differences between the three groups for education, income, employment, marital status, and gender.

In terms of clinical symptoms which did not differ significantly enough to allow distinguishing clinical cases from controls, they found lateral excursions, anterior and posterior occlusion, extent of prior dentistry, presence of prostheses, missing teeth, or chin scars did not aid in distinguishing clinical TMD cases from controls. They concluded that response of pain, pain from palpation, and vertical range of mandibular motion were the most distinguishable characteristics of TMD patients. Many other

signs and symptoms of TMD, such as joint sounds, uncorrected mandibular deviation upon opening, were also as prevalent in the control cases as the TMD patients. They feel TMD is a pain condition in which clinical signs do not necessarily progress to physical deterioration and loss of function. The principal sign, pain, appears to disappear with advancing age. Finally, clinical findings did not vary by age.

In a sample of 637 urban Swedish persons interviewed and examined for clinical signs and symptoms of TMD, 58% had TMJ sounds and 34% of the lateral pterygoid muscles and 27% of the temporalis muscles were tender to palpation (Agerberg et al., 1990). Women were found to report and have these areas of palpation tenderness more than men, and 26% had moderate and severe signs of clinical dysfunction (D_{II} and D_{III}) as compared to only 12% of the men. Only 12% of the sample was free of any signs and symptoms of TMD, while most signs were found to be of mild character (D_I) (69%). The authors feel in part the differing results of this study in terms of the differences found between men and women may be attributable to the greater stress in an urban lifestyle.

A cross-sectional epidemiologic survey to describe the signs and symptoms in an adult sample (over 20 years of age) living in southwest Sweden showed in general there was a dramatic increase in frequency of general diseases and medications beginning at the age of 50 (Salonen et al.,

1990). Below that age about 15-25% of the sample was on medication or had some general disease. Mild to severe subjective symptoms were reported more frequently by 20 to 39 year old women and gradually reversed in the oldest subjects. Approximately 55% of the women and 65% of the men aged 20 to 29 reported no symptoms.

Maximum opening decreased with age as well as lateral and protrusive boarder movements. Of the 20 to 29 year old age group, 99% could open greater than 40 mm, and less than 30 mm was only observed in subjects 70 years or older. No differences between the sexes were found.

TMJ deviation and sounds were found more frequently in women than men at all age groups. No TMJ locking or luxation was found in any of the subjects indicating age is not a factor for TMJ function. Only 2% of the sample had TMJ pain to palpation which was more common in women, and only 0.7% of the sample had pain with movement of the mandible.

Muscle pain was more common in women than in men, however there were no age differences. No sites of muscle pain were found in 82% of 20 to 69 year olds at each age group.

Almost 93% of the subjects were symptom-free or had mild dysfunction ($D_i I$). Symptoms did increase in age, however, the prevalence of $D_i III$ was less than 1%.

Epidemiologic Incidence/Prevalence in Orthodontic Patients

Adult

A retrospective study of 23 adolescent orthodontic patients, between the ages of 24 and 28, a minimum of ten years after treatment, found that the fixed/activator orthodontic treatment cases had fewer TMD symptoms than the general population (Larsson and Ronnerman, 1981). Using the Helkimo Indices, no clinical dysfunction symptoms were found in 65% of the patients, mild in 31%, and only one patient (4%) had symptoms of severe disturbances. The anamnestic dysfunction index showed only 27% of the sample had mild symptoms. The occlusal state index showed 17% with no disturbances, 52% had mild disturbances, and 30% had severe disturbances. Their conclusion was that orthodontic treatment prevents rather than causes functional disturbances.

In a similar retrospective study of 60 Class II, division 1 cases (30 nonextraction and 30 four premolar extraction) five years out of retention compared to an untreated control group (N= 30) found that orthodontic treatment had a favorable result on TMD (Janson and Hasund, 1981). Again using the Helkimo Indices, they found fewer treated cases had moderate and severe clinical dysfunction than the controls. Only 7% of the controls were symptom-free.

A cross sectional study of fixed orthodontic treatment effects on TMD concluded that orthodontic treatment had no effect on TMD (Sadowsky, Begole and Polson, 1980, 1984). The 1980 findings of 75 orthodontically treated patients between the ages of 25 and 55, and the 1984 study of 207 orthodontically treated patients demonstrated that the orthodontically treated group had the same prevalence of TMD signs and symptoms as an untreated control group.

A retrospective study comparing signs and symptoms of craniomandibular disorders between 51 19-year-old orthodontically treated patients (28 girls, 23 boys) with 47 19-year-old nonorthodontically treated controls (19 girls, 28 boys) used all of the Helkimo Indices (Dahl et al., 1988). The orthodontically treated group was on average 5 years out of retention. No significant differences were found between the groups with the exception of the anamnestic portion where nonorthodontically treated groups reported the most symptoms. The orthodontically treated group had 43% with mild clinical dysfunction and 28% with moderate symptoms, compared to 40% and 28% respectively in the control group. The authors concluded that there were no substantial differences between the two groups.

Likewise, in a longitudinal study of the effects of fixed (N=72)/modified functional activator (N=63) orthodontic treatment on TMD, orthodontic treatment neither

helped nor caused TMD (Dibbetts and van der Weele, 1987, 1991). Begg Class I and II treatments did not reduce the percentage of subjective symptoms, incidence of radiographic findings, or objective signs 10 years posttreatment. In general, symptoms tended to increase with age (21% to 41% overall), which concurs with TMD epidemiologic findings. The difference in symptom frequencies between activator and Begg treatment was attributed to age and were no longer present 10 years after treatment.

In the follow-up study, Class I malocclusions always showed the highest CMD frequencies, however age accounted for the different symptom frequencies in treatment modalities (Dibbetts and van der Weele, 1991). Subjective symptoms increased from 20% to 62%. Clicking, which increased from 23% to 36% after four years, was found to be associated with first premolar extraction, however this is possibly due to sampling effects. No other serious symptoms of TMD were related to extractions.

A recent investigation compared 54 former orthodontic patients and to 52 untreated individuals all of whom were 20 to 30 years old (Kess et al., 1991). The orthodontically treated cases were all started in the mixed dentition, 41 with functional appliances and 13 with no fixed appliances. None of the patients had a chief complaint of myofunctional disturbances. The Helkimo Indices were calculated in

addition to other more precise methods of evaluation. The results indicated only 16% of the orthodontically treated group had limitation of movement, and none had pain as compared to 14-17% of the untreated group. In summary, 35% of the treated group was symptom-free as compared to 10% of the untreated. However, 30% of the treated group as compared to 15% had moderate symptoms. Desired lateral guidance, cuspid guidance, occurred 20% more often in the treated group. They concluded that orthodontic treatment has a positive effect on the function of the stomatognathic system, and that there was no connection between functional disturbances and orthodontic treatment.

A recent longitudinal study measuring signs and symptoms of orthodontic patients similar to the Iowa TMD Study is underway at the University of Florida (Hirata et al., 1992). Utilizing a matched control group of untreated patients, the results indicated no change in range of motion over time for either group, no consistent pattern of joint sounds over time. However, joint sounds overall increased from pretreatment levels of 22% to 35% after almost 2 years of orthodontic treatment, however the controls actually had a decrease in frequency of joint sounds from 41% to 31.7%. In both controls as well as orthodontically treated subjects, the prevalence of 3 mm of deviation upon opening or closing increased from the pretreatment level (34%) to

42.1% for the treated group and 51.1% for the controls approximately one year later.

Another mixed longitudinal study of 18 months duration completed at the University of Connecticut concluded that none of the 451 subjects involved developed TMJ signs or symptoms during the course of treatment (Rendell et al., 1992).

Iowa TMD Study

The prospective longitudinal Iowa TMD study (1983 to present) to evaluate the incidence of TMD as a result of, or during orthodontic treatment also has found no relationship between various aspects of orthodontic treatment and TMD (Kremenak et al., 1992, 1992a).

The Iowa TMD Study was started in 1982 by Thomas Melcher (1983) under the direction of Dr. David Kinser. It has employed the Helkimo Dysfunction Index as designed by Marti Helkimo in 1974 for assessing the epidemiological prevalence of TMD in the Lapp population (Helkimo, 1974, 1974a, 1974b, 1974c). The Occlusal Index of the original Helkimo Indices was never implemented.

Melcher's (1984) thesis addressed the question of a significant difference between mean pre- and posttreatment Helkimo scores in a cross sectional design. The results showed significantly less mandibular dysfunction in patients after completion of orthodontic treatment.

Wright (1985) examined the "during treatment" effects of orthodontics and found no significant difference between Helkimo scores, on the same patients, before orthodontic treatment and during treatment.

Harrison (1986) examined the dental and skeletal relationships to determine if there were relationships which would predispose a patient to have TMD problems. Except for possibly class III's, no skeletal or dental relationships were important factors for TMD.

Ziaja (1987) in the first longitudinal study of 31 patients with pre- and posttreatment Helkimo scores, looked for significant associations between factors involving treatment variables and occlusion. He found only class III patients continued to have the highest pretreatment Helkimo scores. In addition, based on longitudinal data, 84% of the cases remained the same or got better from initial to deband, while 16% did increase in Helkimo score.

Harmon (1988) looked at the influence of articular and muscular components of TMD and found that pain to palpation of the lateral pterygoids was found in fewer patients after treatment than before.

In an additional research project, Harmon found no significant difference between the Helkimo Indices scores in debanded orthodontic patients as compared to untreated, matched controls (for age at debanding, sex, and

pretreatment malocclusion) (Kremenak, 1991). She concluded that orthodontic treatment was not an important etiologic factor for TMD.

Ordahl (1989) examined the role of trauma and found that the only factor of importance for increasing Helkimo scores was whiplash. However, the sample was extremely small for a conclusion to be drawn.

Demro (1990) examined the relationship of orthognathic surgery to TMD, and found that at 3 months after surgery 53% of the patients had worse Helkimo scores than pretreatment.

Doleski (1991) continued the mixed longitudinal orthognathic study increasing the sample size and evaluating skeletal relationships, age, type of surgery, length of fixation, and magnitude of surgical movement. He found a significant increase in postsurgical Helkimo scores 3 months after surgery, however by 6 months they returned to pretreatment values. The other variables evaluated had no influence on postsurgical Helkimo scores.

Menard (1991) continued Harmon's other project which involved premolar extraction treatment. There was no significant difference in the deband Helkimo score between premolar extraction treatment, either four bicuspid or two bicuspid, and non-extraction treatment (Kremenak et al., 1992).

Clinical Signs and Symptoms Investigated

The following review will address the frequencies and ranges of the clinical signs to be investigated in this study for both the previous nonpatient samples and orthodontic patient samples. The following values described are in general for the 16 to 30 age group which corresponds to the age range evaluated in the present investigation.

Range of Motion

Once a person reaches maturity, a limited range of mandibular motion rarely exists in the nonpatient populations. Only 1.3% were found to have limited opening of less than 40 mm and only 1.8% could not move laterally greater than 4mm in 222 dental and dental hygiene students (Pullinger et al., 1988a). Deviated opening, which is more closely related to physical TMJ impairment, occurred with a higher frequency (22%) in the students. In a study of 119 twenty year olds, impaired mandibular mobility was found in 11% of the subjects and 24% had deviation of greater than 2mm (Magnusson et al., 1991). Nilner found 50% of the 15 to 18 year old individuals displayed deviation in opening (Nilner, 1981a).

Agerberg and others found a significantly larger opening for men than women, however other border movements were not significantly different (Agerberg et al., 1990). For the entire sample 18 to 64 years old, 55% were without

any impaired mandibular mobility, 36% had slight impairment, and 10% had severe impairment as defined by the Helkimo Indices.

Swanljung and Rantanen (1979) found 6.4% of the 560 Finnish subjects age 18 to 46 had limited opening of 39 mm or less. Deviation of greater than 2 mm was noted in 17.7% of the 583 subjects.

Rieder and others found that a restricted opening of less than 40 mm was found in 5.4% of the sample and in general remained relatively constant throughout all adult age groups until it increased in the over 60 age groups. Deviation upon opening was a more frequent finding, occurring in 25.2% of the sample and decreasing with age from 29.7% in patients less than 30 years old to 21% by 50 to 59 years old (Reider et al., 1983).

In the Lapps, 42% had visible deviations of the mandible during opening and closing movements. Maximal opening averaged 46 ± 7 mm, with lateral excursions averaging almost 20% of this (Helkimo, 1974). The low average may be more related to the small physical stature of the Lapps rather than a true reflection of mandibular mobility dysfunction.

Most (70%) of the persons evaluated had no impairment of mobility as scored by the clinical dysfunction index, 27% had slightly impaired mobility, and 3% had severely impaired

mobility (Helkimo, 1974a). Severe mobility impairment increased with increasing age, while slight impairment remained a relatively constant frequency at 29% from 25 to 65 years of age (Helkimo, 1974c).

Gross and Gale found a mean maximal opening for males of 47.9 ± 7.65 mm and 45.4 ± 5.99 mm for females not including overbite (Gross and Gale, 1983). Others have found the mean maximum opening for males to be 55.1 ± 6.8 mm (Mohlin et al., 1976). The percentage of males and females with less than 37 mm of opening was 5.7% and 7.3% respectively. Within the less than 37 mm group, 20 to 29 year old group was statistically lower, 2.8% of the total.

In addition, they found 1 mm deviation on opening in 17.0% males and 18.3% of the females which was a relatively constant frequency throughout all age groups.

Dworkin and others found that females on average for both TMD affected groups and controls had 4 to 5 mm less maximum vertical opening (Dworkin et al., 1990). Using 35 mm for males and 30 mm for females (millimeters between incisal edges), only 22% of the clinical TMD patients were found to have restricted opening. In all other range of motion measurements, there were no significant interaction between males and females or the groups they were in. Only 13% of the controls had an uncorrected deviation during vertical opening, as opposed to 29% of the clinical cases

and 26% of the community cases. Dworkin also found that the clinical measures of TMD did not change with age, but remained relatively constant. In fact, Dworkin and others found that clinical TMD cases could be assisted in opening on average 10 mm beyond the point of pain. Therefore, there may not be the physical limitations in female patients previously suspected.

Schiffman (1990) and others found maximum opening of less than 40 mm in only 8% of the subjects, and pain on opening in 14%. Interestingly, just the opposite occurred with protrusion with 19% of the subjects being able to protrude less than 7 mm, while 6% experienced pain with this excursion. Right and left laterotrusion movements were less frequently restricted to less than 7 mm 4 to 6%, and less frequently painful, 6 to 9%.

Solberg and others found that only 3.5% of the subjects had interincisal opening of less than 40 mm (Solberg et al., 1979). Deviation of the mandible with opening occurred in 18.3% of the subjects. A 5 mm deviation of the mandible while opening occurred in 9% of the 253 inductees, and irregular movements occurred in 10% (Molin et al., 1976).

For previously treated orthodontic patients, Kess and others found nearly the same percent of orthodontically treated and untreated subjects with no limitation in protrusion, however approximately 50% of the untreated

showed limitation during right and left excursions as compared to 16% of the treated (Kess et al., 1991). This is in close agreement with Larsson and Ronnerman who found 13% had impaired mandibular mobility (Larsson and Ronnerman, 1981).

In another comparison of untreated and orthodontically treated groups, all mean measurements of mandibular movements showed no impairment of mandibular movement (Janson and Hasund, 1981). It is interesting to note that the four premolar extraction group had the smallest mean measurements of movement. In conclusion, orthodontic patients tended to have 2 to 3 times less impaired mandibular function than untreated or normal population subjects.

Temporomandibular Joint Sounds

Temporomandibular joint (TMJ) sounds are the most common finding in epidemiologic studies of nonpatient populations (Wabeke, et al., 1989). In general, clinical TMJ sounds are evenly distributed in both males and females in all adult age groups. Often they are the sole sign or symptom of dysfunction. Wannman and Agerberg found only clicking sounds in 22% of the 285 17-year-olds studied (Wannman and Agerberg, 1986a). Like other signs of TMD they appear to increase in frequency with age from childhood through adolescence. There is no conclusive evidence of TMJ

clicking progressing to a degenerative state (Wabeke et al., 1989; Tallents et al., 1991). A three year longitudinal follow-up study of patients with reciprocal clicking found in 71% of the patients the clicking remained unchanged, in 20% it disappeared with normal opening and without deviation, and in only 9% it degenerated to limited and deviated opening (Lundh et al., 1987).

Nilner found 14% of the 309 15 to 18 year old subjects experienced clicking sounds (Nilner, 1981a). TMJ sounds in 253 military inductees occurred at a frequency of 8% (Mohin et al., 1976). Clicking occurred in 22.7% of the sample of 20 year old Swedes (Magnusson et al., 1991) In a slightly older sample, TMJ sounds were found in 28.3% of the college students examined (Solberg et al., 1979). In a similar study, joint sounds were found in 29% of the students as clicking, while only 3% had crepitus sounds (Pullinger et al., 1988a).

Gross and Gale found on average that joint sounds increased from 0% in the 0- to 9-year-old group to an overall average prevalence of 34.7% beginning with the 20- to 29-year-old group (Gross and Gale, 1983). Females had a significantly higher prevalence of joint sounds (39%) than males (28.5%). Joint sounds did increase to a peak in the 40- to 49-year-old group 43.8% and then declined to a relatively constant level of prevalence of 30% thereafter.

Reider and others found that 49.6% of their private practice sample had joint noises which did increase from less than 30 years of age (40.6%) to approximately 50% by the 30 and older age groups (Reider et al., 1983).

In general population studies, the frequency of sounds ranges from 20 to 70%. Joint sounds occurred in 22.8% of the 583 Finnish subjects studied (Swanljung and Rantanen, 1979). Helkimo found 48% of the Lapps had palpable TMJ sounds although, 17% of these were crepitation sounds (Helkimo, 1974). Over 40% had no TMJ impairment, while 60% had sounds in one or both joints without greater than 2 mm deviation in the path of closure. None of the subjects had severe impairment. When TMJ sounds occurred, they had nearly the same frequency in every age group, that is about 50% in 15- to 44-year-old individuals. However, occurrence increased to 71% in the 45- to 65-year-olds (Helkimo, 1974c).

Agerberg and others found 58% of the urban Swedish sample had TMJ sounds (Agerberg et al., 1990). None of the entire sample had impaired TMJ function, and 60% of the men and 39% of the women were without any impaired TMJ function. Additionally, 40% of the men and 61% of the women had slight impairment.

Dworkin and others identified three types of TMJ sounds: clicking, crepitus, grating in a clinical study

which compared TMD patients to nonpatients (Dworkin et al., 1990). Interestingly, they found the detection of TMJ sounds to be unreliable ($K=0.26$) as compared to digital palpation ($K=0.62$) and considering all of the symptoms measured, joint sounds were the least reliable. In general, 43% of the clinical TMD cases had joint clicking, while 33% of the community cases had it and only 25% of the controls. Crepitus was equally distributed among all three groups at 8%. Thus as in previous studies, joint sounds were the most common sign, however as stated earlier, the clinical significance has not been determined.

For previously treated orthodontic patients, Kess and others found no significant differences between orthodontically treated and untreated subjects with regard to clicking during opening or closing. There was a 6% significant difference in intermedial closing sounds of the untreated as compared to the treated (13.5% verses 7.4%) (Kess et al., 1991).

Larsson and Ronnerman found clicking to be the most common TMD sign present in postorthodontically treated patients (1981). Likewise, based on anamnestic findings, TMJ sounds were the most frequent symptoms for both treated and control groups (Janson and Hasund, 1981).

Based on clinical examination, untreated controls had greater impaired TMJ function than treated groups. Of the

orthodontically treated groups, 26.7% were symptom free compared to 10%, 61.7% had slight TMJ impairment verses 70%, and severe TMJ impairment was noted in 11.7% of the treated groups verses 20% of the untreated. Clicking and crepitation prevalence was found to increase from 23% pretreatment to 36% four years later which remained relatively unchanged for the next 10 years (Dibbets and van der Weele, 1991).

In the University of Illinois study of TMD and orthodontic treatment, 34.4% of the treated group had clinically palpable TMJ sounds compared to 41.8% of the controls (Sadowsky and BeGole, 1980). For a similar study at Eastman Dental Center, the results found were 32.4% and 28.8% respectively (Sadowsky and Polson, 1984).

In a cross-sectional study of TMJ sounds in 347 orthodontic pretreatment, during treatment, and posttreatment, the frequency of joint sounds were 40.8%, 60.8%, and 56.8% respectively (Sadowsky et al., 1985). Associations were found between joint sounds, age, and treatment, however, they could not state whether the increase in sounds were due to age or orthodontic treatment. No associations were found between TMJ sounds and dental wear as well as a number of functional occlusal factors. In a later study, the prevalence of sounds before orthodontic treatment occurred in 36.3% of adults and adolescents (Runge

et al., 1989). The results did not indicate a correlation between TMJ sounds and malocclusions.

In a longitudinal study evaluating the changes in joint sounds with orthodontic treatment, 160 patients who ranged in age from 9 to 41 (mean age of 14.5 years) were found to undergo a decrease in clinically identifiable joint sounds after orthodontic treatment (Sadowsky *et al.*, 1991). Initially, 25% of the subjects had joint sounds which decreased to 16.2% after treatment. The only significant association was that older age groups tended to have a higher pretreatment frequency of sounds.

Pain

Temporomandibular Joint

Palpation

Pain from the TMJ area or joint itself during function or palpation is perhaps the most important diagnostic sign of TMD. It is the major reason for which patients seek TMD therapy. In general, general joint symptoms increased with age, while 15- to 24-year-old subjects had the fewest frequency of symptoms (Helkimo, 1974c). Understandably, one would expect the frequency of subjective TMJ pain to correlate well with the prevalence of patients who have actually been treated for TMD. Dworkin and others found lateral palpation of the TMJ in TMD patients to be painful in 56.9% of the cases as opposed to 9.1% of the controls,

and intrameatal palpation to be tender in 10.2% versus 2.9% (Dworkin et al., 1990). In general, lateral TMJ pain palpation was about five times more common than palpation pain via the external meatus.

Lateral TMJ palpation pain occurred in 7% of 253 Swedish inductees, usually unilateral (Molin et al., 1976). In a similar age group, lateral TMJ tenderness to palpation was found in 9.9% of the 285 17-year-old subjects, and 2.8% had tenderness to posterior palpation (Wannman and Agerberg, 1986a). Solberg and others found TMJ pain upon palpation in 5.3% of the subjects (Solberg et al., 1979). In a TMD study of young dental students, joint pain was found in only 14% of the subjects, the remainder were asymptomatic, 13% of which was mild to moderate (Pullinger et al., 1988). Gross and Gale found lateral TMJ tenderness to palpation in 3.9% of the subjects and posterior tenderness in 0.7% (Gross and Gale, 1983). A majority of the tenderness was of a mild nature. Pain was found upon lateral and posterior palpation in 1.9% of the 583 Finnish subjects (Swanljung and Rantanen, 1979). Pain to TMJ palpation was found in 3.4% of the 20-year-olds (Magnusson et al., 1991).

Higher prevalences are reported by other studies. Nilner (1981) found 34% of the young adults (15 to 18 years old) examined to have pain from palpation of the TMJ, 22% posterior, and 23% lateral. Rieder and others found TMJ

pain from palpation in 31.6% of the sample. Its frequency decreased gradually with age from 38.8% in the less than 30-year-old-group to 23.2% in the over 60 age group (Rieder et al., 1983). Helkimo found 45% of the subjects had pain in the TMJ as a result of palpation, 24% of which were by external meatus palpation, while only 21% felt TMJ pain only when it was palpated laterally (Helkimo, 1974). The distribution of palpable TMJ pain was about equal (about 24%) for all age groups (Helkimo, 1974c).

Schiffman and others found that pain to lateral palpation of the TMJ capsule occurred in about 25% of the subjects, while posterior palpation pain occurred in about 19% of the subjects (Schiffman et al., 1990). Agerberg and others found palpable TMJ tenderness laterally in 16% and posteriorly in 3% of the men and 15.2% and 13.3% respectively of the women age 18 to 64 (Agerberg et al., 1990). In other words, 81% of the men and 72% of the females were symptom free.

Comparing orthodontically treated subjects to untreated subjects, 81.5% versus 63.5% of the untreated had no tenderness during lateral palpation of the TMJ during opening (Kess, et al., 1991). In a similar comparison, 93.3% of the orthodontically treated subjects were without TMJ pain, while only 73.3% of the untreated were (Janson and Hasund, 1981). Posterior palpation pain was present in 1.6%

of the orthodontically treated subjects and 3.3% of the untreated. Larsson and Ronnerman found no orthodontically treated patient had palpable TMJ pain (1981).

Function

Similar to TMJ pain, pain with mandibular function had a low frequency in the nonpatient populations studied. Only 12 (5%) of 222 dental and dental hygiene students had pain with maximum opening, 1 with protrusion, and 3 with left laterotrusion had pain associated with these movements (Pullinger et al., 1988). Wannman and Agerberg (1986a) found no incidence of pain with mandibular movement in the 285 17-year-olds studied. Schiffman and others found maximum opening pain in 14% of the subjects (Schiffman et al., 1990).

Pain with mandibular opening was reported significantly more frequently in clinical TMD cases (55 to 46.5%) than community cases (31.2 to 25.2%) or controls (11.2 to 7.8%) for lateral and protrusive movements (Dworkin et al., 1990).

In an urban Swedish population, pain was found in one movement of the mandible in only 3% of the sample, and the other 97% were completely free of pain in all movements included in the Helkimo clinical dysfunction index (Agerberg and Inkapool, 1990). Of 583 Finnish subjects, 4.0% had pain with maximal mandibular movements (Swanljung and Rantanen, 1979). Pain on mandibular movement was found in 3.4% of the

20-year-olds (Magnusson et al., 1991). Nilner found only 10% of the 15- to 18-year-old subjects experienced pain upon opening of the mouth (Nilner, 1981a).

Helkimo found 33% of the sample experienced pain with mandibular movement (50% in one movement, and 50% in more than one movement) (Helkimo, 1974). In this group, 21% experienced pain with maximum opening, followed with lesser frequency in right and left excursions, protrusion, closing, and retrusion. The TMJ was the most common area of this pain (65%), followed by the cheek and throat.

Dworkin and others (1990) found pain on function in about 50% of the clinical TMD cases, 21% of the community cases, and 10% of the control cases (Dworkin et al., 1990). Almost half of the TMD patients localized the pain to the joint or muscles of mastication in combination, while less than 2% of the controls had TMJ pain in the joint.

Comparing an orthodontically treated sample to an untreated sample, no former patient was found to have pain with maximum opening or protrusion, and less than 4% had pain with lateral excursions (Kess et al., 1991). The untreated sample, 14% had pain with maximum opening, 17% with protrusion, and 15% with right lateral movement. Larsson and Ronnerman found no orthodontically treated subject with pain during mandibular movements (Larsson and Ronnerman, 1981).

Combining both four premolar extraction and nonextraction orthodontic treatment groups, 66.7% were free of pain on movement of the mandible as compared to only 33.3% of the untreated group, 15% versus 20% had slight impairment, and 18.3% versus 46.7% had severe impairment as determined by the Helkimo Indices (Janson and Hasund, 1981).

Masticatory Muscles

Masticatory muscle pain to palpation appears to be a relatively common epidemiologic finding, although some have found it to have a high specificity and sensitivity for TMD (Cacchiotti et al., 1991). About 50% of the muscle sites respond with pain, with one site in five likely to be severely tender (Solberg, 1982). Masticatory muscle tenderness to palpation occurred in 13% of the 253 inductees (Molin et al., 1976). In a similar age group, 27% had one to three sites of muscle tenderness, and 15% had four or more (Wannman and Agerberg, 1986a). Solberg found pain with masticatory muscle palpation in 34.2% of the subjects (Solberg et al., 1979). Muscle tenderness was found in 38.7% of the 20 year-old Swedish subjects (Magnusson et al., 1991).

The most common muscle tender to palpation found was the lateral pterygoid (37.9%) in 285 17-year-old subjects, and 23.9% had tenderness to the temporal insertion palpation (Wannman and Agerberg, 1986a). Nilner (1981) found 55% of

the 309 15- to 18-year-old subjects experienced tenderness to muscle palpation with the lateral pterygoid and temporalis showing a 27% and 20% frequency respectively.

Agerberg found palpable muscle tenderness most frequently in the lateral pterygoid (34.5%) and temporalis insertion (27.2%) (Agerberg *et al.*, 1990). In terms of Helkimo's clinical dysfunction index, 63.4% of the men and 46.1% of the women were free of masticatory muscle pain from palpation. More than four sites were found in 2.5% of the men and 5.9% of the women, while one to three sites were found in 34.1% of the men and 48% of the women.

Pullinger and others found 48% of the subjects reported at least one area of muscle tenderness to palpation (Pullinger *et al.*, 1988a). In this group was 13% with moderate tenderness and 19% with severe tenderness. The most common sites for moderate to severe were the lateral pterygoid (37%), masseter (23%), and the temporal muscle tendon (17%).

In the Lapps, masticatory muscle pain to palpation occurred in 66% of the sample (Helkimo, 1974). The most common area found in 70% of those with muscle pain, was with the attachment of the temporalis muscle. Other frequent areas of tenderness to palpation include: the lateral pterygoid (59%), masseter (40%), anterior temporalis (34%) and the posterior belly of the digastric muscle (30%).

Absence to tenderness from masticatory muscle palpation was noted least often (50%) in the 15- to 24-year-olds. In general, the remaining age groups had equal frequencies of sites of tenderness. Pain from muscle palpation was found on average in 15.5% of the sample. Specifically, it decreased with age from 23.7% in less than 30 years old subjects to 9.3% in the 50 to 59 age group (Rieder et al., 1983).

Gross and Gale found that 14.6% of patents age 20 to 29 had tenderness to palpation of the lateral pterygoid area with approximately three pounds of pressure for two seconds (Gross and Gale, 1983). In addition, they found 0.6% from the anterior temporalis, 0.7% from the posterior temporalis, 2.4% from the masseter, and 2.5% form the medial pterygoid. In general, an increase in prevalence with age was noted.

Dworkin and others found that use of three pounds of pressure for muscle palpation was unjustified because it caused overestimation of muscle pain. Therefore, they used 1 pound of force (Dworkin et al., 1990). Their findings indicate that clinical TMD cases always had the highest prevalence of palpable muscle pain. The masseter was painful in 53.6% of the TMD cases as opposed to 11.1% of the controls; the anterior temporalis was 32.1 versus 5.7%; posterior temporalis 17.1 versus 1.9%; tendon of the

temporalis 74.2 versus 40.9%; lateral pterygoid 74.3 versus 44.7%.

Cacchiotti and others studied signs and symptoms in patients with and without TMD (Cacchiotti et al., 1991). The mean age groups for both groups was 25.2 years in the patient group and 24.9 in the control group. Questionnaires, clinical exam, and diagnostic casts (mounted in centric relation) were used to investigate the differences. They found only four factors were statistically significant between the two groups: 63% of the TMD patients had frequent headaches while only 33% of the normals did. TMD patients had an average of 2.6 tender masticatory muscles while the normals were next to none (0.2). Likewise, cervical muscle tenderness was also very specific for TMD patients (0.97). Maximum opening was significantly restricted in the patient group averaging 42.9 mm while the controls averaged 49.8 mm.

Schiffman and others found the lateral pterygoid was most frequently painful to palpation (53%) (Schiffman et al., 1990). In descending order from there was the anterior temporalis (40%), the masseter (40%), medial pterygoid (12%), and the temporalis insertion (9%).

Comparing orthodontically treated subjects with untreated controls, no tenderness to palpation of any masticatory muscle was found in 64.8% versus 40.4% of the

controls (Kess et al., 1991). In the treated group, 5.6% of the anterior temporalis, 11.1% of the masseter, and 14.1% of the lateral pterygoids were painful to palpation. In a similar comparison, 58.3% of orthodontically treated subjects verses 36.7% of untreated individuals were free of any muscle pain (Janson and Hasund, 1981). One to three tender sites were found in 33.3% of the treated group, but in 50% of the untreated group. Finally, four or more palpable sites were found in 8.3% of the treated group and in over 13% of the untreated. Larsson and Ronnerman found only one of 23 (4%) postorthodontically treated patients to have pain with palpation of the muscles of mastication (Larsson and Ronnerman, 1981).

Inter-examiner Reliability of Clinical Measurements

In an evaluation of three different methods of TMD evaluation, it was found that the oral history taken by the examiner was the most unreliable (Rieder, 1977). A written self-administered questionnaire such as the Helkimo anamnestic form was found more reliable. The most reliable method of assessment of TMD signs was found to be direct clinical examination.

In a study of the reproducibility of five answers from a TMD questionnaire after one week by patients with masticatory dysfunction, the range of reproducibility was 90

to 71.5% (Kopp, 1976). The poorest reproducibility was for TMJ and masticatory muscle pain. The greatest amount of agreement between the questionnaire and the clinical exam involved joint sounds. There was no agreement between the questionnaire and the exam of the masticatory muscles. The author felt this was primarily due to the variable nature of pain, and the patients lack of ability to discriminate between muscle and joint pain.

Dworkin expressed his concern over the lack of examiner reliability in studies largely due to the lack of training, lack of specific, defined criteria, and variability of signs and symptoms independent of examiner reliability (Dworkin et al., 1990a). Published data supports his view that training and calibration of examiners does improve examiner reliability (Dworkin et al., 1988). However, these same results indicate low reliability for subjective evaluation of joint sounds and palpation of the masticatory muscles and TMJ. Understandably, the highest reliability occurred in the clinical measurements which involved measuring instruments like rulers.

Certainly, ideal criteria for defining TMD would leave no room for subjective interpretation by the examiner or subject. At present the signs and symptoms utilized in epidemiologic studies need considerable amounts of recognition by the patient and evaluation by the examiner.

This subjectivity compromises comparisons of studies due to examiner error which is inherent with different observers (Solberg, 1982).

Mohlin and others in a study of 1018 12-year-olds evaluated examiner variability by repeating random examinations on the same day (Mohlin et al., 1991). Using Cohen's kappa, they found high reproducibility between opening and protrusive movements. All other variables included in the Helkimo clinical dysfunction index showed substantial to an almost perfect degree of agreement. Measurement of centric relation and centric occlusion were the most variable and therefore, not recommended as longitudinal study variables.

Carlsson found better agreement between examiners when differentiating between individuals with clinical signs of dysfunction and those without (Carlsson et al., 1980). However, intra-observer symptom measurements and palpation measurements over time in a longitudinal study had as great a variability as inter-observer variation largely due to symptom variation. Classification of sounds and mandibular positions were highly variable. As a result, he recommended that longitudinal measurements should be performed by the same person and with the use of controls.

In general, results from questionnaires correspond well to clinical findings if the questions are clinically

relevant. Reproducibility and validity, if related to clinical findings are satisfactory (Kopp, 1976; Reider, 1977). However lack of agreement on a standard format makes comparisons of studies difficult if not impossible.

Dworkin's experience with reliability in epidemiologic TMD studies indicates that training examiners to specific, well defined criteria is imperative in order to produce reliable epidemiologic TMD studies (Dworkin et al., 1990a; 1990b). They found the greatest reliability with clinical measurements involving measuring instruments and the lowest with clinical measurements involving examiner assessment, especially anterior occlusal classification, joint sounds, and muscle palpations. Reliability was found to increase when combining several individual measurement scores, such as a computed index measure, although they mask the reliability of measuring a particular component (Dworkin et al., 1990).

Part of this problem with reliability of measurement is the variability of signs and symptoms, a characteristic of TMD. The challenge then is to distinguish between the biologic change and the unreliability in measuring. It has been known for a long time that clinical measurements in medicine have poor inter-examiner reliability (Koran, 1975). Therefore training and calibration of examiners are a

necessary consideration of longitudinal studies of this nature.

In what is likely the most relevant study of intra- and interobserver variability of the evaluation of the signs and symptoms making up the Helkimo index, 19 subjects ranging in age from 21 to 29 years were randomly examined twice by two examiners over a three hour period (Kopp and Wenneberg, 1983). All subjects were free of symptoms of TMD. In addition to the areas used by the Helkimo clinical dysfunction index, the retruded and intercuspal positions were recorded. The results indicated that maximum opening had the lowest intra- and interobserver variability and was therefore the most reliable sign evaluated. Additionally, the intra-observer variability of the clinical dysfunction index agreed on average 66% of the time, while the clinical dysfunction scores agreed only 53% of the time and muscle palpation observations were in agreement only 53% of the time on average.

Inter-observer assessment of clinical dysfunction index agreed on average only 61%, with the actual clinical dysfunction score in agreement between the two observers only 32% of the time. Surprisingly, muscle palpations agreed on average 58% of the time. Based on these results, Kopp and Wenneberg recommended not utilizing multiple

observers for the investigation of the signs used to derive the Helkimo clinical dysfunction index.

These results are a strong contrast to the level of agreement found in an earlier study in which interobserver agreement of the clinical dysfunction index over the course of a year was found to be 73% in adults (Carlsson et al., 1980). In spite of this result, they also concluded that longitudinal studies of TMD would minimize observer error if the same observer did the examinations and control groups were used.

The percent agreement found for each of the components of the Helkimo clinical dysfunction index are as follows: mandibular mobility 83%, TMJ function 70%, muscle pain 80%, TMJ pain 93%, and mandibular movement pain 93%. The initial observer agreement of 73% for the clinical dysfunction index fell to 54% after one year. They felt variations in recorded clinical signs come from natural changes, poor precision or reproducibility of the methods, difference in examination technique, and differences in opinions regarding positive and negative findings.

Summary

Clinical research is often sharply criticized for a number of reasons. Mainly, it is considered less precise than lab experiments due to the greater difficulties involved in controlling extraneous variables and obtaining a

random, homogeneous sample. Often these difficulties interfere with the quality of the results (Carlsson *et al.*, 1980).

Certainly these criticisms are valid when evaluating the available scientific studies of TMD. Prevalence differences could be due to variations in: interpretation of results, examination technique and observers, questionnaire wording and response alternatives. In addition to these limitations, there is the fluctuate nature of TMD. Thomson found 12 to 20% of his nonpatient control group with signs of TMD disappeared spontaneously after one year (Thomson, 1971). In cross sectional studies, it is not possible to establish a cause and effect relationship, nor the estimation of strength of the risk factor-symptom relationships with measures such as odds ratios and attributable risk (Locker and Slade, 1988).

Inter- and intra-reliability studies clearly emphasize the importance of defining the measured criteria, training and evaluating the examiners, and the need to avoid changes in methods during longitudinal studies.

In spite of these limitations, it is evident that a number of trends and ranges for the prevalence of TMD in nonpatient and orthodontic patients, as well as the prevalences of the signs and symptoms of TMD can be determined.

In general, most epidemiologic TMD studies have found the occurrence of at least one sign of TMD to range from 41 to 77% in the adult population (Agerberg and Carlsson, 1972; Helkimo, 1972, 1974, 1974a, 1974b, 1974c; Ingervall et al., 1974; Molin et al., 1976; Williamson, 1977; Osterberg et al., 1979; Swanljung and Rantanen, 1979; Nilner, 1981, 1981a; Rugh et al., 1985; Solberg, 1979, 1982, 1985, 1987; Dworkin et al., 1990; Schiffman et al., 1990).

Overall, a comparison of studies show the range of frequency of subjective symptoms to vary from 25% to 58% (Agerberg and Carlsson, 1972; Helkimo and Zarb, 1979; Swanljung and Rantanen, 1979).

Children 6 to 15 years of age appear to have a lower incidence (30 to 60%) of clinical signs than others, but the prevalence increases as children get older. In general, signs and symptoms increase with age up to 15 years, and then remain at a relatively constant prevalence or increase throughout all age groups thereafter (Egermark-Eriksson, 1981; Solberg, 1987).

Typically, 12 to 20% of the adolescent and adult populations have no signs or symptoms of TMD, while 12 to 88% have at least one sign of TMD which is usually subclinical. Of this group, 12 to 59% are aware of some TMD symptoms, and of these people, only 4 to 7% would warrant definitive TMD treatment.

Most epidemiologic studies of the general population indicate that the incidence of TMD is about the same for men and women, and relatively constant throughout all adult age groups. On the other hand, females tend to seek medical treatment more frequently, therefore, they tend to present for TMD treatment three to nine times more frequently than men (McNeill, 1990a).

One possible explanation for women having greater frequency of TMD signs, but the same frequency of symptoms, is that the signs are not specific etiologic factors for TMD (Solberg, 1979). Higher prevalence of posterior TMJ palpation tenderness may be related to the fact that women are more prone to develop musculoskeletal diseases, for example, osteoarthritis and rheumatoid arthritis (Solberg et al, 1979; Agerberg, 1990).

In general though, only 5 to 7% of the symptomatic group have symptoms severe enough to warrant treatment. The importance of the urban 20 to 40 year old age range is because these are the people who most frequently seek TMD treatment (Clark and Mulligan, 1984; Rugh and Solberg, 1985).

It can be assumed that orthodontic patients prior to treatment would have the same or greater prevalence of TMD dysfunction as the general population. Therefore, one should expect 30 to 66% of the children and adolescents and

approximately 32 to 88% of the adults to have at least one sign of TMD. This high prevalence in the potential pre-orthodontic population underscores our need to have a thorough understanding of this disorder in order to properly manage these patients.

The clinical implications of the signs reviewed indicate: joint sounds, of the clicking and popping nature, alone are not good indicators of TMD due to the high frequency of occurrence in the nonpatient population, and the fact that it is frequently the sole sign or symptom. None of the reviewed studies suggested joint sounds would warrant treatment per say. Epidemiologic studies of clinically determined joint sounds have reported their frequency ranging from 0% in children less than 9 years old to about 50% in people over 30 years old. In general, subjects 15 to 30 years of age have an increase in frequency from 14% to 41%, respectively.

This can be compared to the 7.4% to 36% range stated for adolescent and adult orthodontic patients.

In general the next most common sign reported after TMJ sounds was muscle tenderness/fatigue. In general, the reported range of frequency of this finding in the adolescent and adult populations was 13% to 66%. The most common muscles of mastication tender to palpation were the lateral pterygoid (frequency range of 27 to 59%), temporalis

(frequency range 17 to 70%), and the masseter (frequency range 11 to 40%).

Orthodontic patients reported masticatory muscle pain to palpation in the range of 4 to 42%.

Pain is a key clinical sign of TMD because it is the major reason patients seek treatment (Solberg, 1979). Only 3 to 34% of the joints examined in epidemiologic studies were actually painful. Frequently, this sign was accompanied by other signs and symptoms of TMD. Crepitation was an even rarer finding, being found in only 1 to 15% of the general population.

Two to 19% of orthodontic patients reported TMJ pain after completion of treatment.

Pain on mandibular movement was likewise an infrequent finding occurring in up to 33% of the general population.

Similar to the general population, orthodontic patients reported pain with mandibular movement in range of 0 to 18.3%.

Deviation with opening in the general population occurred in a range from 9 to 50%, depending on the tolerance of deviation. Limited range of motion was a very infrequent finding occurring in just 1 to 22% of the general population.

Orthodontic patients reported a limited range of in 0 to 16% of the subjects, while deviation occurred in 30 to 50% of the subjects.

The presence of more than one of the cardinal symptoms (Bell, 1990) or more than one of the accepted signs and symptoms used in most of the epidemiologic indices, would warrant further evaluation for possible TMD treatment. However at this time, it is still unclear which of these signs and symptoms are absolutely necessary for TMD to be considered present, progressing, or in need of treatment.

A high percent of potential orthodontic patients, at one time or another, have experienced at least one sign or symptom of TMD (26 to 76%). The clinical significance of such an occurrence is not yet clear. Therefore, it behooves the orthodontist to remain current with respect to guidelines as to when referral of the patient for a comprehensive TMD evaluation is warranted (McNeill, 1990).

Table 1 Continued

Study	Percent Limited Opening	HELKIMO INDEX SCORE					Symptoms		Reported	
		Dysfunction Q	I	II	III	Anamnestic I	TMJ Sounds	TMJ Muscle	Pain Mn.	Decreased Mov't. Opening
Mohlin, 1991	1	54								
Nilner, 1981										
Egermark-Eriksson 1981	2	64	30	5	1		7			8
	1	55	38	6	1		11			6
	0	38	48	12	2		21			5
1992		25	52	23						
Wannman, 1986		44	42	15	0	80	13	7	13	1.4
Solberg, 1979	3.5									1.8
Ingervall, 1980		39	33	25	2					
Schiffman, 1987	1.2	7	34	33	26	39	17	44		
Agerberg, 1972										
Helkimo, 1974	0	12	41	25	22	43	31	26	35	39
Gross, 1983	6.5									7
Rieder, 1983	5.4									8
Pullinger, 1988	1.3	40	41	17	1	20				14.1
Magnusson, 1991	11	51			1					
Solonen, 1990	5	46	47	1				13		
Agerberg, 1990		12	69	17	2					
Locker, 1988						58	23	19	25.4	12.9
									21.4	
										7
									2.7	
										1.4
										7.4
ORTHODONTICALLY TREATED PATIENTS										
Egermark-Eriksson 1992		68	25	8						
Dahl, 1988		29	43	28	0	29	71	0		
Kess, 1991		35	34	25	4					
Larsson, 1981		65	31	4		73	27	0		
Janson, 1981		16	46	26	13	57	22	20		

METHODS AND MATERIALS

Longitudinal Sample

One way to determine whether orthodontic treatment factors are of importance or not, would be to follow patients from beginning of treatment until the end of treatment or retention. Comparing their incidence of TMD signs and symptoms with a normal incidence may allow conclusions to be drawn about the effects of orthodontics on TMD. It was in this framework that the Iowa longitudinal TMD study was started to evaluate the effects of orthodontic treatment on TMD.

All subjects in the study are initially selected based on age, 16 to 30 years of age, and availability from the graduate clinical patient pool at the end of the summer semester. Each year the study is continued, a new group of approximately 30 patients are examined before beginning orthodontic treatment to establish a pretreatment Helkimo score.

Ideally, the patients are to have an examination pretreatment, during treatment on a yearly basis, at deband, and then once a year through retention until dismissal.

Figure 1

Flowchart of Longitudinal Iowa TMD Study 1984-1992.

Melcher's Original Study	Wright's Follow-up Study	Harrison's Follow-up Study	Ziaja's Follow-up Study	Harman's Follow-up Study	Ordahl Follow-up Study
Group 1 Pretreatment N= 29	Group 3 Pretreatment N= 28	Group 6 Pretreatment N= 29	Group 10 Pretreatment N= 36	Group 15 Pretreatment N= 35	Group 21 Pretreatment N= 24
Group 2 Posttreatment N= 30	Group 4 In treatment N= 28	Group 7 In treatment N= 23	Group 11 In treatment + Posttreatment N= 25	Group 16 In treatment + Posttreatment N= 31	Group 22 In treatment + Posttreatment N= 31
	Group 5 Posttreatment N= 26	Group 8 In treatment N= 26	Group 12 In treatment + Posttreatment N= 23	Group 17 In treatment + Posttreatment N= 21	Group 23 In treatment + Posttreatment N= 23
		Group 9 Posttreatment N= 18	Group 13 In treatment + Posttreatment N= 20	Group 18 In treatment + Posttreatment N= 14	Group 24 In treatment + Posttreatment N= 11
			Group 14 Posttreatment N= 11	Group 19 In treatment + Posttreatment N= 13	Group 25 In treatment + Posttreatment N= 6
				Group 20 Posttreatment N= 8	Group 26 In treatment + Posttreatment N= 9
					Group 27 Posttreatment N=2

Figure 1 Continued

Demro's Follow-up Study	Doleski's Follow-up Study	Kharouf's Follow-up Study
Group 28 Pretreatment N= 30	Group 36 Pretreatment N= 20	Group 45 Pretreatment N= 13
Group 29 In treatment + Posttreatment N= 22	Group 37 In treatment + Posttreatment N= 4	Group 46 In treatment + Posttreatment N= 16
Group 30 In treatment + Posttreatment N= 16	Group 38 In treatment + Posttreatment N= 9	Group 47 In treatment + Posttreatment N= 16
Group 31 In treatment + Posttreatment N= 16	Group 39 In treatment + Posttreatment N= 10	Group 48 In treatment + Posttreatment N= 7
Group 32 In treatment + Posttreatment N= 12	Group 40 In treatment + Posttreatment N= 14	Group 49 In treatment + Posttreatment N= 5
Group 33 In treatment + Posttreatment N= 4	Group 41 In treatment + Posttreatment N= 6	Group 50 In treatment + Posttreatment N= 8
Group 34 In treatment + Posttreatment N= 8	Group 42 In treatment + Posttreatment N= 4	Group 51 In treatment + Posttreatment N= 5
Group 35 Posttreatment N= 2	Group 43 In treatment + Posttreatment N= 8	Group 52 In treatment + Posttreatment N= 0
	Group 44 Posttreatment N= 1	Group 53 In treatment + Posttreatment N= 1
		Group 54 Posttreatment N= 0

Present Sample

Presently, there are over 300 patients recorded in the Iowa TMD Study since the inception in 1983, Figure 1. The sample for this study, Table 2, consisted of patients from the initial groups to the present patient groups, who met the following criteria: pretreatment score, during treatment score, and a posttreatment score within 13 months of the deband date. There were 95 patients who met this criteria by January 15, 1992. All subjects in the present sample were used to determine subjective and objective findings.

Table 2

The Number of Subjects in the Present Sample Under Study
From Each of the Original Pretreatment Groups of the
Longitudinal Study.

Pretreatment Group	Examiner	Number of Subjects
Group 1	Melcher	19
Group 3	Wright	18
Group 6	Harrison	14
Group 10	Ziaja	18
Group 15	Harman	13
Group 21	Ordahl	11
Group 28	Demro	0
Group 36	Doleski/Menard	2
Group 45	Hull/Kharouf	0
Total Sample Size		95

Reliability Sample

The reliability sample of 12 subjects was a convenience sample randomly drawn from students, staff and faculty to

assess the reliability of the Helkimo clinical dysfunction examination technique. In general, they tended to be older in age than the present sample.

Technique for Assessment of Temporomandibular Dysfunction

The Helkimo Examination Technique

The technique used to determine the Helkimo score was the same as that described by Melcher (1984) and Helkimo (1974). The only changes made to the original form as outlined by Melcher were the addition of a cover page to inform the patient in writing about the study, the addition of a verbal question to elicit whether the subject had taken any medications within the past 24 hours, and typographical format changes which aided data entry.

Patients were initially verbally invited to participate in the study during a regularly scheduled appointment. If they agreed to participate, then a written summary of the research project was presented to the subject (APPENDIX A pg. 176). The questionnaire and examination was completed at this appointment.

The Helkimo examination form, as used in this study, is presented in APPENDIX A. In Melcher's (1983) following description, new reference and APPENDIX A citations are placed within brackets.

Format of the Anamnestic and Clinical Indices

Each subject was initially required to sign a consent form to participate in the study, and each was also asked to complete a personal history form, so they could be contacted for further follow-up studies [APPENDIX A, pgs. 177 and 178].

Mandibular dysfunction within the groups was assessed by a combination of questionnaire and clinical examination. A standardized form was used for each, patterned after the system originally described by Helkimo (1974a,b,c) [APPENDIX A, pgs. 179 - 181].

This system was chosen because of the documented reliability between observers and fairly high correlation between objective and subjective findings (Helkimo, 1974a). Previous tests have shown the index to be both valid and reliable (Helkimo, 1974b; Nilner, 1981; Molin et al., 1976; Egermark-Eriksson et al., 1981).

These standardized indices allow signs and symptoms to be numerically weighted according to severity, and a total sum of findings can be calculated, which allows placement of a given subject into a particular dysfunction group. This is done for both the anamnestic and clinical indices (Helkimo, 1974a).

Method of Assessment

After consenting to be a subject in the Iowa TMD study, the subject was asked to complete the personal history portion of the form (APPENDIX A, pg. 178). If they were undergoing subsequent examinations, the patient was only asked to update information which may have changed. On the bottom of the form, the examiner would note what phase of treatment the patient was in and verbally ask if the patient had taken any medication within the previous 24 hours. If

the patient gave a positive response, then the medication, dosage, and frequency was noted.

They were then instructed to answer each question "yes" or "no" (APPENDIX A, pg. 179). Questions numbered 1 and 2 had additional responses requested if they were initially answered positively.

The format for the clinical exam followed the standardized form demonstrated in [APPENDIX A, pgs. 180-182]. The Angle classification, as determined by buccal segment relationships (cuspid relationship), was noted for each patient. Overbite was measured by vertical overlap of the central incisors, and overjet taken as the distance between the labial surface of the mandibular central incisors and the lingual surface of the maxillary central incisors at the labial edge. All measurements were made with a millimeter ruler and were expressed to the nearest one-half millimeter. Any variation between right and left incisors was resolved by taking the average of the two measurements.

A line scribed on the mandibular incisors aided in measurement of the overbite and the mandibular movements. Deviation upon opening and closing of 2 mm was estimated with the naked eye.

Mandibular mobility was assessed by measurements of the range of movement. A millimeter ruler was again used to find the maximum opening, as measured at the incisal edges, to which the overbite measurement was added. Lateral movements were measured by using the dental midlines as a reference point. Any midline discrepancy was resolved by placement of a pencil line on the lower incisor surface, which represented a corrected midline. Protrusion ability was based on the distance from the incisal edge of the maxillary incisor to the labial surface of the mandibular incisor, to which the overjet measurement was added. After each movement was tallied, a sum, which represented the mandibular mobility index, was

obtained and entered into Part A of the clinical dysfunction index [APPENDIX A pg. 180].

The patients were then instructed to open and close several times while observed both from the front and from behind, to detect any deviation on opening. Bilateral joint palpation with index fingers was used to feel and hear any joint noises, and Part B was scored.

Masticatory muscle palpation was performed by bilateral multidigit palpation extraorally. Intraorally the index finger was used to palpate bilaterally. Each muscle site indicated on [APPENDIX A pg. 182] was checked by several palpations at various locations within the muscle belly. Any muscle tenderness or pain was contrasted between right and left sides and charted as positive. The sum of symptomatic sites was entered in Part C [APPENDIX A pg. 181].

TMJ pain palpation was bilateral as well as muscles except for lateral pterygoid and temporalis insertion which were palpated unilaterally. Subjects were asked to differentiate between discomfort of the palpation and pain from the muscle. If pain was the response, then it was recorded as muscle tenderness.

Temporomandibular joint pain was evaluated by lateral pressure exerted on the joints with the index fingers, and placement of the small fingers into the external auditory meatus anteriorly. Subjects were instructed to open and close with pressure in both positions and any pain or tenderness was recorded. Pain upon anterior palpation of the auditory meatus is usually indicative of posterior capsulitis and is weighed as being more severe than lateral pain. Part D was thus completed in this manner.

Finally, the patient was asked to complete the movements performed during assessment of mandibular mobility and then asked how many, if any, of the movements caused pain or discomfort in the TM joints or facial musculature. This completed the scoring of Part E and the Clinical Dysfunction Index. A sum total could then be obtained to indicate numerically the dysfunction group, as indicated on

the bottom of the Clinical Dysfunction Index [APPENDIX A pg. 181] This total was used as the total dysfunction score.

After completion of the data collection, patients were informed they would be given subsequent TMD examinations on an annual basis. The findings were entered into a computer for statistical analysis. In addition, each subject was assigned a patient ID number and placed into a group corresponding to the year of initiation into the study.

Method of Assessment of the Reliability Sample

The reliability sample tended to be older than the actual study sample because it was a convenience sample of the available faculty, staff, and students. The twelve subjects in the reliability group were examined using the same method as used on the sample subjects, but independently by other investigators all in one day. This method of calibration allows for comparisons of reliability between examiners.

Statistical Analysis

Analysis of Reliability Data

Inter-examiner reliability was assessed from data collected by each of the three investigators (C. Menard, N. Hull, J. Kharouf) for the mandibular mobility indices, the clinical dysfunction index, and the masticatory muscle palpation exam form (APPENDIX A pg. 180-182) on a separate group of 12 subjects.

Cohen's Kappa statistics were calculated for parametric and non-parametric variables of the Helkimo Indices between the two present examiners for both calibration periods.

Analysis of Longitudinal Data

Analysis of the Influence of Orthodontic Treatment on the Anamnestic Data

The frequency distribution of positive responses for the entire sample was determined for the initial anamnestic data, that is, collected before treatment began, during treatment, posttreatment, and into retention.

Analysis of the Influence of Orthodontic Treatment on Clinical Dysfunction Data

Descriptive statistics were used to define the sample and subgroups. Analysis of the five components of the clinical dysfunction index of the Helkimo Indices was carried out by compiling the frequency distributions of clinical symptoms according to the degree of severity into "3x3", and "9x3" tables. The data were then evaluated using the nonparametric "sign test". The level of significance, $p \leq 0.05$, of the finding was determined by comparing the chance of occurrence from a binomial table to a probability of 0.5 for the null hypothesis of symmetric discordance.

RESULTS

Inter-examiner Reliability Study

The results of the Cohen's Kappa statistics of the initial calibration (26 March 1991) on 12 subjects showed the greatest differences between the present two examiners existed in the measurement of overbite and overjet (Table 3). The effects of recording measurements as an index can best be seen by comparing the higher agreement found in the

Table 3.

Assessment of Inter-examiner Variability of Parametric and Non-parametric Variables of the Helkimo Clinical Dysfunction Indices Between Examiners I and II

Variable	N	Initial Calibration Kappa	Recalibration Kappa
Angle Class	12	1.00	1.00
Overbite	12	0.67	0.80
Overjet	12	0.67	1.00
Mobility			
Opening	12	0.91	1.00
Right	12	0.91	1.00
Left	12	0.91	1.00
Protrusion	12	1.00	1.00
Mobility Index	12	1.00	1.00
TMJ Function	12	0.91	0.91
Muscle Pain	12	1.00	1.00
TMJ Pain	12	1.00	1.00
Movement Pain	12	1.00	1.00
Helkimo Score	12	0.98	0.98

components of the mandibular mobility index with overbite and overjet. Linear measurements which are indexed allowed the examiners to increase their agreement to 91%.

All of the variables were within an acceptable range of inter-examiner reliability after initial calibration.

Inter-examiner reliability was reassessed from clinical dysfunction indices data collected by four examiners during recalibration one year (19 March 1992) after initial calibration (Table 3). Results of the Cohen's Kappa calculated for the same two initial examiners on 12 different subjects indicated that the two examiners had remained reliable one year later. In addition, Cohen's Kappa statistics on the nonparametric variables (mobility index and clinical dysfunction index) for both years suggest that the Helkimo index is a reliable index over time.

The results from these calibrations indicate that Helkimo Indices scores for nonparametric variables obtained by one examiner did not differ greatly from scores obtained by the second examiner. Second, the reliability study suggests that the intra-examiner clinical examination technique did not differ greatly over one year.

Pretreatment Descriptive Statistics of the Sample

Patient Attributes

The distribution of the sample by pretreatment age is presented in Table 4. The ages ranged from 15 to 35 years with a mean of 19.4 years and with the mode occurring at 16 years of age. Almost 98% of the sample ranged in age from 15 to 25 years of age which was the initial age range selected by Melcher (1983) who began the Iowa TMD Study. Females comprised 66.3% (63) of the sample, while males comprised 33.7% (32). The ratio of females to males varied considerably for each Angle class.

Table 4

Frequency and Percentage Distribution of Pretreatment Age in the Sample

Age (Years)	Frequency	Percent	Cumulative Percent
15	9	9.5	9.5
16	24	25.3	34.7
17	11	11.6	46.3
18	4	4.2	50.5
19	6	6.3	56.8
20	5	5.3	62.1
21	8	8.4	70.5
22	4	4.2	74.7
23	6	6.3	81.1
24	6	6.3	87.4
25	10	10.5	97.9
26	1	1.1	98.9
35	1	1.1	100.0
N=95		Mean= 19.4 \pm 3.8 years	

In terms of actual malocclusions represented in the sample, Table 5 shows that 58% of the sample had one form or another of an Angle class II malocclusion. The other large portion of the sample was comprised of Angle class I malocclusions (36.8%). Class III malocclusions continue to

Table 5

Frequency and Percentage Distribution of Pretreatment Angle Classification in the Sample

Angle Class	Male/Female	Total Frequency	Percent
I	14/21	35	36.8
II			
-1	13/29	42	44.3
-2	3/10	13	13.7
III	2/3	5	5.3
Total		95	100.0

be the rarest subjects, representing only 5.3% of the present sample. These figures approximate the general prevalences of the types of Angle malocclusions found in the nonpatient population.

The mean pretreatment overbite was 3.5 mm. Table 6 shows 95.6% of the overbites ranged from 0 to 7 mm. Pretreatment, 35.8% of the subjects had an overbite in the range of 2 to 3.5 mm, which can be considered an orthodontic treatment goal range. Overjet had a mean of 2.8 mm which

Table 6
Frequency and Percentage Distribution of Pretreatment
Overbite in the Sample

Overbite (mm)	Frequency	Percent	Cumulative Percent
-2	1	1.1	1.1
-1	1	1.1	2.1
0	3	3.2	5.3
0.5	4	4.2	9.5
1	6	6.3	15.8
1.5	1	1.1	16.8
2	8	8.4	25.3
2.5	3	3.2	28.4
3	14	14.7	43.2
3.5	9	9.5	52.6
4	17	17.9	70.5
4.5	2	2.1	72.6
5	13	13.7	86.3
5.5	1	1.1	87.4
6	8	8.4	95.8
7	2	2.1	97.9
8	1	1.1	98.9
9	1	1.1	100.0
N= 95 Mean = 3.5 ± 1.9 mm			

would also be considered a treatment goal at the completion of active orthodontic treatment. Table 7 shows 94.6% of the pretreatment overjets fell within the range of 0.5 to 7 mm.

Anterior and/or posterior crossbites of any tooth or teeth, occurred in 20 (21.1%) of the subjects. These were either single teeth, whole segments, or complete (scissor) crossbites occurring uni- or bilaterally.

Table 7
Frequency and Percentage Distribution of Pretreatment
Overjet in the Sample

Overjet (mm)	Frequency	Percent	Cumulative Percent
-1	1	1.1	1.1
0	2	2.1	3.2
0.5	11	11.6	14.7
1	13	13.7	28.4
1.5	4	4.2	32.6
2	10	10.5	43.2
2.5	10	10.5	53.7
3	10	10.5	64.2
3.5	8	8.4	72.6
4	8	8.4	81.1
4.5	3	3.2	84.2
5	7	7.4	91.6
6	2	2.1	93.7
6.5	1	1.1	94.7
7	3	3.2	97.9
8	1	1.1	98.9
10	1	1.1	100.0
N= 95		Mean = 2.8 \pm 2.0 mm	

Functionally, none of the subjects in this sample had any limited maximum opening (less than 40 mm) pretreatment. Only 6 (6.3%) subjects had slight impairment in their right mandibular lateral excursion, and only 9 (9.5%) had slight impairment when moving to their left (Table 8).

The greatest frequency of restriction in mandibular movement occurred with protrusion of the mandible with 14.7% of the subjects exhibiting slight impairment, but only 1 subject being severely impaired pretreatment (Table 9).

Table 8

Frequency and Percentage Distribution of Pretreatment
Maximum Mandibular Lateral Movements in the Sample

Maximum Right Laterotrusion	Frequency	Percent
≥ 7 mm	89	93.7
4-6 mm	6	6.3
Maximum Left Laterotrusion		
≥ 7 mm	86	90.5
4-6 mm	9	9.5

Table 9

Frequency and Percentage Distribution of Pretreatment
Maximum Mandibular Protrusion in the Sample

Maximum Protrusion	Frequency	Percent
≥ 7 mm	80	84.2
4-6 mm	14	14.7
0-3 mm	1	1.1

Treatment Modalities

The following modalities were employed during treatment of the present sample. Table 10 shows that 24.2% of the subjects had no extractions involved with their treatment, while nearly as many (25.3%) had four bicuspids removed in any combination. The largest group of extractions was the miscellaneous group which included three subjects who just

had third molar removal for orthodontic treatment and three who had third molar removal in combination with some other extraction pattern.

Table 10
Frequency and Percentage Distribution of Extractions in the Sample

Extraction Pattern	Frequency	Percent	Cumulative Percent
0 : No Extractions	23	24.2	24.2
1 : Any Four Bicuspids	24	25.3	49.5
2 : Any Two Upper Bicuspids	16	16.8	66.3
3 : Any Other Extraction Pattern	32	33.7	100.0

Headgear of any type including J-hook was used in 49.5% of the cases (Table 11). No attempt was made to account for time used, quantify force or direction, or estimate actual compliance.

Table 11
Frequency and Percentage Distribution of Headgear Therapy in the Sample

Headgear Use	Frequency	Percent	Cumulative Percent
None	48	50.5	50.5
Used	47	49.5	100.0

Class II elastics were also used with a high frequency in 72.6% of the subjects (Table 12). Like headgear, the only criteria for a positive finding was their use for at least one month.

Table 12

Frequency and Percentage Distribution of Class II Elastic Therapy in the Sample

Class II Elastic Use	Frequency	Percent	Cumulative Percent
None	26	27.4	27.4
Used	69	72.6	100.0

As expected, class III elastics were used in only 10 patients (Table 13). This sample had 5 Angle class III subjects.

Table 13

Frequency and Percentage Distribution of Class III Elastic Therapy in the Sample

Class III Elastic Use	Frequency	Percent	Cumulative Percent
None	85	89.5	89.5
Used	10	10.5	100.0

Treatment Results

Finally, the mean treatment time for the present sample from the initial visit until debanding was 23.1 months (Table 14). The range in treatment for 96.8% of the subjects was 12 to 35 months. Long treatment times do not

Table 14

Frequency and Percentage Distribution of Length of Treatment in the Sample

Months in Treatment	Frequency	Percent	Cumulative Percent
12	2	2.1	2.1
13	3	3.2	5.3
14	1	1.1	6.3
15	7	7.4	13.7
16	6	6.3	20.0
17	1	1.1	21.1
18	4	4.2	25.3
19	8	8.4	33.7
20	6	6.3	40.0
21	12	12.6	52.6
22	4	4.2	56.8
23	5	5.3	62.1
24	4	4.2	66.3
25	3	3.2	69.5
26	4	4.2	73.7
27	1	1.1	74.7
28	4	4.2	78.9
29	2	2.1	81.1
30	1	1.1	82.1
31	4	4.2	86.3
32	1	1.1	87.4
33	4	4.2	91.6
34	4	4.2	95.8
35	1	1.1	96.8
44	2	2.1	98.9
51	1	1.1	100.0
N= 95		Mean = 23.1 \pm 7.3 months	

necessarily represent difficult treatments and should not suggest difficult treatment in terms of TMD symptoms. Rather treatment length is more likely a result of a combination of other factors such as cooperation and orthopedic treatment objectives.

Pretreatment Clinical Dysfunction Index Distribution

The frequency of the five signs which comprise the clinical dysfunction index of the Helkimo indices are given in Tables 15 through 18.

Table 15, distribution of the mandibular mobility index, is directly related to the mandibular movements ranges given in Tables 8 and 9. Almost 79% of the sample had no impaired mandibular range of movement. The 20% with a slight impairment were the subjects who had limitations in right and left lateral movements or protrusion. The one subject with a severe impairment was the subject with the

Table 15

Frequency and Percentage Distribution of Pretreatment
Impaired Range of Mandibular Movement/Mobility Index (A) in
the Sample

Degree of Impairment	Frequency	Percent	Cumulative Percent
0 : None	75	78.9	78.9
1 : Slight	19	20.0	98.9
5 : Severe	1	1.1	100.0

severe limitation in protrusion from Table 9.

Table 16 shows that joint sounds and/or mandibular deviation less than or equal to 2 mm on opening or closing was the most frequent sign in the present sample. The prevalence, 38.9%, is within the range found in other epidemiologic studies of the prevalence of TMD signs and symptoms cited in the literature review. Only 1 subject had severely impaired TMJ function prior to orthodontic treatment.

Table 16

Frequency and Percentage Distribution of Pretreatment Impaired TMJ Function (B) in the Sample

Degree of Impairment	Frequency	Percent	Cumulative Percent
0 : None	57	60.0	60.0
1 : Sounds/Deviation	37	38.9	98.9
5 : Locking	1	1.1	100.0

Masticatory muscle pain to palpation, of 6 pairs of muscles, was the second most common TMD sign, Table 17. This sample's frequency, like TMJ sounds, corresponds well with the ranges reported in the literature review. Muscle pain occurred in 31.6% of the subjects, with 8.4% having four or more areas and thus being scored as having a severe muscle impairment.

The muscles noted to have the highest frequency of tenderness where the right and left lateral pterygoid (total of 23) , followed by the medial pterygoid (total of 21), and finally, the temporalis insertion (total of 16). No pain was elicited from the posterior temporalis muscles and very infrequently from the anterior temporalis muscle. Pain from masseter palpation occurred in between the frequencies of these two groups (total of 11).

Table 17

Frequency and Percentage Distribution of Pretreatment Muscle Pain (C) in the Sample

Sites Tender to Palpation	Frequency	Percent	Cumulative Percent
0 : None	65	68.4	68.4
1 : 1 - 3	22	23.2	91.6
5 : 4 or More	8	8.4	100.0

Actual joint pain from palpation occurred in 11.6% of the subjects pretreatment, with almost twice the frequency from posterior palpation, Table 18. Over 88% of the sample had an absence of joint pain.

Table 18

Frequency and Percentage Distribution of Pretreatment TMJ Pain (D) in the Sample

Tenderness to Palpation	Frequency	Percent	Cumulative Percent
0 : None	84	88.4	88.4
1 : Lateral	4	4.2	92.6
5 : Posterior	7	7.4	100.0

Pain with one or more of four mandibular border movements, maximum opening, right and left lateroexcursions, and maximum protrusion, occurred in 7.4% of the sample pretreatment, Table 19.

Table 19

Frequency and Percentage Distribution of Pretreatment Pain on Movement of the Mandible (E) in the Sample

Pain on Movement	Frequency	Percent	Cumulative Percent
0 : None	88	92.6	92.6
1 : One Movement	5	5.3	97.9
5 : Two or More Movements	2	2.1	100.0

Table 20 shows that the during treatment score was taken most consistently approximately one year after treatment began. The reason for this is most likely due to the fact that active patients tend to make monthly

appointments, therefore the recall problem which exists for the collection of posttreatment scores does not exist during treatment. Patients were selected, as stated in the methods and materials section, if they had a during treatment score and also if the posttreatment score occurred within 13 months after treatment. One subject whose posttreatment score was beyond 13 months (a sample selection criteria) was included in the sample. Without this subject the mean time for Helkimo score collection from deband to posttreatment is 6.5 ± 3.8 months with a range of 0 to 13.5 months.

Table 20 suggests that the time of collection of the pretreatment as well as during treatment scores was not influenced by the severity of those scores. The standard deviation of the during treatment scores was only 1.5 months. Posttreatment scores of up to 13 months after

Table 20
Time Between Collection of Helkimo Scores

Time Interval	Mean	S. D. [Months]	Range
Pretreatment to During Treatment	12.4	1.5	7.9-16.8
During Treatment to Posttreatment	16.8	7.4	4.6-41.9
Pretreatment to Posttreatment	29.2	7.5	14.4-53.4
Posttreatment to Posttreatment II	14.6	8.5	2.1-42.6
Deband Date to Posttreatment	6.7	4.0	0.0-21.6

treatment is an appropriate length of time because most patients utilize retainers which are usually worn full time for the first year. This retention wear, while not active treatment, does in some patients prolong occlusal settling which is a desired immediate posttreatment goal.

Heterogeneity of Sample's Pretreatment Attributes

The following tables give further detailed, baseline description of the sample pretreatment. The Tables 21-23 define what types of patients comprised the sample, and demonstrate the lack of sample homogeneity across pretreatment conditions and treatment modalities. Finally, Tables 24-28 display the lack of homogeneity across the pretreatment clinical dysfunction signs.

Table 21 shows the distribution of the sample across the four patient attributes evaluated. Overbite and overjet were divided less than or equal to 3 mm and 3.5 mm or greater. Consideration was given to dividing these two patient attributes into three divisions, however this would have further increased the sparsity of the sample. Crossbite, as stated earlier, was either present (yes) or not present (no).

The type of subjects in greatest frequency (18.9%) were Angle Class II with deep bites, large overjets, and no crossbites. Following this subgroup of subjects in terms of frequency were: Angle Class II deep bites, small overjets

Table 21

Frequency and Percentage Distribution of the Pretreatment
Patient Attributes

Angle Class	Overbite	Overjet	Crossbite	<u>Subjects</u>	
				N	Percent
I	≤3.0	≤3.0	No	8	8.4
I	≤3.0	≤3.0	Yes	5	5.3
I	≤3.0	≥3.5	No	6	6.3
I	≤3.0	≥3.5	Yes	0	0
I	≥3.5	≤3.0	No	12	12.6
I	≥3.5	≤3.0	Yes	2	2.1
I	≥3.5	≥3.5	No	2	2.1
I	≥3.5	≥3.5	Yes	0	0
II	≤3.0	≤3.0	No	10	10.5
II	≤3.0	≤3.0	Yes	2	2.1
II	≤3.0	≥3.5	No	4	4.2
II	≤3.0	≥3.5	Yes	2	2.1
II	≥3.5	≤3.0	No	13	13.7
II	≥3.5	≤3.0	Yes	4	4.2
II	≥3.5	≥3.5	No	18	18.9
II	≥3.5	≥3.5	Yes	2	2.1
III	≤3.0	≤3.0	No	2	2.1
III	≤3.0	≤3.0	Yes	2	2.1
III	≤3.0	≥3.5	No	0	0
III	≤3.0	≥3.5	Yes	0	0
III	≥3.5	≤3.0	No	0	0
III	≥3.5	≤3.0	Yes	1	1.1
III	≥3.5	≥3.5	No	0	0
III	≥3.5	≥3.5	Yes	0	0
Total				95	100.0

and no crossbites (13.7%); Angle Class I (12.6%) deep bites, small overjets and no crossbites, and Angle Class II (10.5%) small overbite, small overjet and no crossbites. These four subgroups of subjects, none of which had crossbites, made up 55.7% of the sample. The remaining 42 subjects were widely distributed across the other possible combinations of Angle

class, overbite, overjet and presence of a crossbite in subgroups of 8 or less. No patients were found in seven of the possible combinations.

Table 21 demonstrates the lack of an adequate sample size when distributed across pretreatment conditions which would be necessary for the evaluation of patient attributes as possible risk factors.

A number of important distinctions between the different Angle Classifications are apparent. Crossbites were found in 18 to 20% of Angle Class I and II patients, but occurred in 60% (N=3) of the Angle Class III patients. Large overjets were characteristically found in 47.3% of the Angle Class II patients, but in just 20% of the Angle Class I patients, and not at all in Angle Class III patients. Likewise, deep bites (≥ 3.5 mm) were found in only 20% of the Angle Class III patients, 45.7% of the Angle Class I patients, and over 67% of the Angle Class II patients.

Heterogeneity of Treatment Modalities

Table 22 shows that the problem of a small sample and increased heterogeneity when the treatment modalities are distributed across pretreatment patient attributes evaluated. The complete lack of homogeneity across the treatment modalities can be seen by the fact that not all Angle Class I, II, or III subjects within those respective classifications receive the same types of mechanical

Table 22

Frequency and Percentage Distribution of Treatment Modalities Utilized Across Pretreatment Attributes

Angle Class	Over-bite	Over-jet	Cross-bite	N	Headgear Pct(N)	Elastics	
						II	III
I	≤3.0	≤3.0	No	8	50.0(4)	75.0(6)	25.0(2)
I	≤3.0	≤3.0	Yes	5	20.0(1)	60.0(3)	0
I	≤3.0	≥3.5	No	6	83.3(5)	100.0(6)	0
I	≤3.0	≥3.5	Yes	0	0	0	0
I	≥3.5	≤3.0	No	12	16.7(2)	33.3(4)	8.3(1)
I	≥3.5	≤3.0	Yes	2	50.0(1)	100.0(2)	0
I	≥3.5	≥3.5	No	2	50.0(1)	0	0
I	≥3.5	≥3.5	Yes	0	0	0	0
II	≤3.0	≤3.0	No	10	50.0(5)	90.0(9)	10.0(1)
II	≤3.0	≤3.0	Yes	2	100.0(2)	50.0(1)	0
II	≤3.0	≥3.5	No	4	75.0(3)	50.0(2)	0
II	≤3.0	≥3.5	Yes	2	50.0(1)	50.0(1)	50.0(1)
II	≥3.5	≤3.0	No	13	38.5(5)	92.3(12)	7.7(1)
II	≥3.5	≤3.0	Yes	4	50.0(2)	75.0(3)	0
II	≥3.5	≥3.5	No	18	83.3(15)	100.0(18)	0
II	≥3.5	≥3.5	Yes	2	0	50.0(1)	50.0(1)
III	≤3.0	≤3.0	No	2	0	50.0(1)	50.0(1)
III	≤3.0	≤3.0	Yes	2	0	0	50.0(1)
III	≤3.0	≥3.5	No	0	0	0	0
III	≤3.0	≥3.5	Yes	0	0	0	0
III	≥3.5	≤3.0	No	0	0	0	0
III	≥3.5	≤3.0	Yes	1	0	0	100.0(1)
III	≥3.5	≥3.5	No	0	0	0	0
III	≥3.5	≥3.5	Yes	0	0	0	0
Total				95	47	69	10

therapy. The reason is not only due to different pretreatment conditions shown in Table 21 which largely dictate specific treatment plans, and therefore mechanotherapy, but also due to the varied treatment approaches of the providers.

Of the four subgroups of 10 or more subjects with similar patient attributes in Table 21, only two subgroups maintain adequate size when including the headgear and elastic used during treatment, see Table 22. Of the largest subgroup from Table 21, Angle Class II deep bite, large overjet, and no crossbite, 15 used headgear, and class II elastics, but not class III elastics. There are also 12 subjects who were Angle Class II, deep bite, small overjet, and no crossbites, who used class II elastics and did not use class III elastics.

In addition, great differences in treatment modalities used for various subgroups emphasizes the difficulty of comparisons and evaluation of posttreatment impairment between these groups. None of the Angle Class III patients used headgear, while 40% of the Angle Class I patients and 60% of the Angle Class II patients did. Class III elastics were used on 80% of the Angle Class III patients, but by only 8.6% of the Angle Class I and 7.3% of the Angle Class II patients. Class II elastics were used on 60% of the Angle Class I patients, 85.5% of the Angle Class II patients, and only 20% of the Angle Class III patients.

When the final treatment modality, extraction pattern, is included there are no subgroups of adequate size left to statistically evaluate (Table 23). The largest subgroup from Tables 21 and 22 is reduced to 8 subjects who are Angle

Class II with a deep overbite, large overjet, no crossbite, used class II elastics but not class III, had two upper bicuspid extracted and had a mean treatment time of 23.3

Table 23

Frequency and Percentage Distribution of Extraction Patterns Across Pretreatment Patient Attributes

Angle Class	Over bite	Over jet	Cross bite	N	Extraction Pattern			
					Percent (N)			
					0	1	2	3
I	≤3.0	≤3.0	No	8	37.5(4)	50.0(4)	0	12.5(1)
I	≤3.0	≤3.0	Yes	5	60.0(3)	0	0	40.0(2)
I	≤3.0	≥3.5	No	6	16.7(1)	83.3(5)	0	0
I	≤3.0	≥3.5	Yes	0	0	0	0	0
I	≥3.5	≤3.0	No	12	41.7(5)	25.0(3)	0	33.3(4)
I	≥3.5	≤3.0	Yes	2	0	0	0	100.0(2)
I	≥3.5	≥3.5	No	2	0	50.0(1)	0	50.0(1)
I	≥3.5	≥3.5	Yes	0	0	0	0	0
II	≤3.0	≤3.0	No	10	0	20.0(2)	20.0(2)	60.0(6)
II	≤3.0	≤3.0	Yes	2	0	0	0	100.0(2)
II	≤3.0	≥3.5	No	4	50.0(2)	25.0(1)	0	25.0(1)
II	≤3.0	≥3.5	Yes	2	0	0	100.0(2)	0
II	≥3.5	≤3.0	No	13	15.4(2)	15.4(2)	23.1(3)	46.2(6)
II	≥3.5	≤3.0	Yes	4	25.0(1)	0	25.0(1)	50.0(2)
II	≥3.5	≥3.5	No	18	2.2(4)	16.7(3)	44.4(8)	16.7(3)
II	≥3.5	≥3.5	Yes	2	0	0	0	100.0(2)
III	≤3.0	≤3.0	No	2	0	100.0(2)	0	0
III	≤3.0	≤3.0	Yes	2	100.0(2)	0	0	0
III	≤3.0	≥3.5	No	0	0	0	0	0
III	≤3.0	≥3.5	Yes	0	0	0	0	0
III	≥3.5	≤3.0	No	0	0	0	0	0
III	≥3.5	≤3.0	Yes	1	0	100.0(1)	0	0
III	≥3.5	≥3.5	No	0	0	0	0	0
III	≥3.5	≥3.5	Yes	0	0	0	0	0
Total				95	23	24	16	32

0: No Extractions

1: Any Combination of Four Bicuspid

2: Any combination of Two Upper Bicuspid

3: Any Other Extraction Pattern

months. Of this eight, 7 worn headgear as a treatment modality. In addition, several of the other larger subgroups of subjects identified from the previous Tables 21 and 22, had large portions of the subgroup fall into extraction pattern 3, the miscellaneous extraction pattern.

It is important to note that large differences existed between the extraction patterns for each Angle Classification. Nonextraction treatment occurred in approximately 40% of Angle Class I and III patients, but in only 16.4% of the Angle Class II patients. In addition, all upper two bicuspid extractions occurred in Angle Class II patients.

Heterogeneity of the Severity or Pretreatment
Signs of the Clinical Dysfunction Index
Across Pretreatment Attributes

In addition, the sample was evaluated for homogeneity across the scores or severity of the pretreatment signs in the five components of the clinical dysfunction index with respect to four of the patient attributes.

Table 24 which displays the mandibular mobility index severity, shows that all 18 of the subjects of the largest subgroup of Angle Class II patients with common patient attributes started without any mandibular mobility impairment. Of the second largest subgroup of Angle Class II patients with common patient attributes, 10 subjects (76.9) started without any mobility impairment. Again, the

first row showing 1 subject with a severe impairment was the subject with limited protrusion from Table 9.

With the exception of the 18 subjects who started out Angle Class II with deep overbite, large overjets and no crossbites, this table, as well as the four to follow, show

Table 24

Frequency and Percentage Distribution of the Pretreatment Mandibular Mobility Index (A) Across Pretreatment Attributes

Angle Class	Over bite	Over jet	Cross bite	N	Mobility Index		
					Pct(N)		
					0	1	5
I	≤3.0	≤3.0	No	8	75.0(6)	12.5(1)	12.5(1)
I	≤3.0	≤3.0	Yes	5	80.0(4)	20.0(1)	0
I	≤3.0	≥3.5	No	6	100.0(6)	0	0
I	≤3.0	≥3.5	Yes	0	0	0	0
I	≥3.5	≤3.0	No	12	75.0(9)	25.0(3)	0
I	≥3.5	≤3.0	Yes	2	50.0(1)	50.0(1)	0
I	≥3.5	≥3.5	No	2	100.0(2)	0	0
I	≥3.5	≥3.5	Yes	0	0	0	0
II	≤3.0	≤3.0	No	10	80.0(8)	20.0(2)	0
II	≤3.0	≤3.0	Yes	2	100.0(2)	0	0
II	≤3.0	≥3.5	No	4	75.0(3)	25.0(1)	0
II	≤3.0	≥3.5	Yes	2	50.0(1)	50.0(1)	0
II	≥3.5	≤3.0	No	13	76.9(10)	23.1(3)	0
II	≥3.5	≤3.0	Yes	2	50.0(2)	50.0(2)	0
II	≥3.5	≥3.5	No	18	100.0(18)	0	0
II	≥3.5	≥3.5	Yes	2	100.0(2)	0	0
III	≤3.0	≤3.0	No	2	0	100.0(2)	0
III	≤3.0	≤3.0	Yes	2	50.0(1)	50.0(1)	0
III	≤3.0	≥3.5	No	0	0	0	0
III	≤3.0	≥3.5	Yes	0	0	0	0
III	≥3.5	≤3.0	No	0	0	0	0
III	≥3.5	≤3.0	Yes	0	0	100.0(1)	0
III	≥3.5	≥3.5	No	0	0	0	0
III	≥3.5	≥3.5	Yes	0	0	0	0
Total				95	75	19	1

the lack of homogeneity in the sample across the severity of the pretreatment signs.

Subjects with crossbites were more likely (almost twice as many, 35.0% versus 16.0%) to experience slight impairment of mandibular mobility, than subjects without crossbites. Overbite appeared to have no influence on mandibular mobility. Subjects with overjets of 3.5 mm or more had less mandibular mobility impairment (94.1% had none), while 29.5% of all subjects with a overjet of less than or equal to 3 mm had slight to severe impairment. This follows with the pattern of the five Angle Class III subjects, 4 of whom had slight mandibular mobility impairment. Angle Class I or II had no effect on mandibular mobility.

Table 25 displays the distribution of the TMJ function impairment across the same four the patient attributes as Tables 21-24. In this table, the largest subgroup, the Angle Class II with deep bites, large overjets, and no crossbites has only 61.1% (N= 11) of the subjects beginning treatment without any TMJ function impairment. Almost 40% of this group had some level of TMJ impairment, including the 1 subject of the entire pretreatment sample with a severe TMJ impairment. This table shows the lack of homogeneity across the severity of TMJ impairment.

As with mandibular mobility, crossbites do affect the frequency of TMJ function impairment although not as

Table 25

Frequency and Percentage Distribution of the Pretreatment
TMJ Function (B) Across Pretreatment Attributes

Angle Class	Overbite bite	Over jet	Cross bite	N	TMJ Function Percent(N)		
					0	1	5
I	≤3.0	≤3.0	No	8	75.0(6)	25.0(2)	0
I	≤3.0	≤3.0	Yes	5	80.0(4)	20.0(1)	0
I	≤3.0	≥3.5	No	6	33.3(2)	66.7(4)	0
I	≤3.0	≥3.5	Yes	0	0	0	0
I	≥3.5	≤3.0	No	12	75.0(9)	25.0(3)	0
I	≥3.5	≤3.0	Yes	2	0	100.0(2)	0
I	≥3.5	≥3.5	No	2	0	100.0(2)	0
I	≥3.5	≥3.5	Yes	0	0	0	0
II	≤3.0	≤3.0	No	10	80.0(8)	20.0(2)	0
II	≤3.0	≤3.0	Yes	2	0	100.0(2)	0
II	≤3.0	≥3.5	No	4	50.0(2)	50.0(2)	0
II	≤3.0	≥3.5	Yes	2	50.0(1)	50.0(1)	0
II	≥3.5	≤3.0	No	13	61.5(8)	38.5(5)	0
II	≥3.5	≤3.0	Yes	4	50.0(2)	50.0(2)	0
II	≥3.5	≥3.5	No	18	61.1(11)	33.3(6)	5.6(1)
II	≥3.5	≥3.5	Yes	2	50.0(1)	50.0(1)	0
III	≤3.0	≤3.0	No	2	50.0(1)	50.0(1)	0
III	≤3.0	≤3.0	Yes	2	50.0(1)	50.0(1)	0
III	≤3.0	≥3.5	No	0	0	0	0
III	≤3.0	≥3.5	Yes	0	0	0	0
III	≥3.5	≤3.0	No	0	0	0	0
III	≥3.5	≤3.0	Yes	1	100.0(1)	0	0
III	≥3.5	≥3.5	No	0	0	0	0
III	≥3.5	≥3.5	Yes	0	0	0	0
Total				95	57	37	1

pronounced. Almost 63% of all subjects without crossbites were asymptomatic for impaired TMJ function, compared to only 50.0% of those with crossbites. As with mandibular mobility, the amount of overbite and Angle Class, including Angle Class III, did not appear to have any association with impaired TMJ function.

More subjects (N= 40) with 3 mm or less overjet had asymptomatic TMJ function than those with 3.5 mm or greater (N= 17). Combining subjects with an overjet of 3.5 mm or greater who had slight and severe (one subject) impairment, 50% of these subjects had TMJ function impairment. This is the opposite association overjet had with mandibular mobility.

Table 26 shows the distribution of muscle pain across patient attributes. The largest subgroup of Angle Class II has the same distribution of severity as with the TMJ function, 61.1% of this group had no muscle pain before beginning treatment. Comparing the distributions of these 18 subjects for TMJ impairment (Table 25) and muscle pain (Table 26), 9 of the 11 asymptomatic subjects were the same in both signs, 4 of the 6 slight impairments were the same, and none of the severe were the same.

Combining every subject by Angle Class, a decreasing trend in the number of muscle sites asymptomatic to palpation was found for Angle Classification. Of the Angle Class I's, 77.1% were asymptomatic, but only 63.6% of the Angle Class II's and 60.0% of the Angle Class III's were asymptomatic. In addition, Class III subjects had the greatest frequency of severe muscle impairment, 40%.

Crossbite did not appear to be associated with muscle impairment. Subjects with overbites of less than or equal

Table 26

Frequency and Percentage Distribution of the Pretreatment
Muscle Pain (C) Across Pretreatment Attributes

Angle Class	Over bite	Over jet	Cross bite	N	Muscle Pain Percent(N)		
					0	1	5
I	≤3.0	≤3.0	No	8	87.5(7)	12.5(1)	0
I	≤3.0	≤3.0	Yes	5	100.0(5)	0	0
I	≤3.0	≥3.5	No	6	66.7(4)	16.7(1)	16.7(1)
I	≤3.0	≥3.5	Yes	0	0	0	0
I	≥3.5	≤3.0	No	12	75.0(9)	25.0(3)	0
I	≥3.5	≤3.0	Yes	2	100.0(2)	0	0
I	≥3.5	≥3.5	No	2	100.0(2)	0	0
I	≥3.5	≥3.5	Yes	0	0	0	0
II	≤3.0	≤3.0	No	10	90.0(9)	10.0(1)	0
II	≤3.0	≤3.0	Yes	2	0	100.0(2)	0
II	≤3.0	≥3.5	No	4	75.0(3)	0	25.0(1)
II	≤3.0	≥3.5	Yes	2	50.0(1)	50.0(1)	0
II	≥3.5	≤3.0	No	13	46.2(6)	30.8(4)	23.1(3)
II	≥3.5	≤3.0	Yes	4	75.0(3)	25.0(1)	0
II	≥3.5	≥3.5	No	18	61.1(11)	33.3(6)	5.6(1)
II	≥3.5	≥3.5	Yes	2	100.0(2)	0	0
III	≤3.0	≤3.0	No	2	50.0(1)	0	50.0(1)
III	≤3.0	≤3.0	Yes	2	50.0(1)	0	50.0(1)
III	≤3.0	≥3.5	No	0	0	0	0
III	≤3.0	≥3.5	Yes	0	0	0	0
III	≥3.5	≤3.0	No	0	0	0	0
III	≥3.5	≤3.0	Yes	1	100.0(1)	0	0
III	≥3.5	≥3.5	No	0	0	0	0
III	≥3.5	≥3.5	Yes	0	0	0	0
Total				95	67	20	8

to 3 mm had decreased numbers of painful muscle sites (75.6%) than subjects with 3.5 mm overbites or larger (63.0%). Likewise, subjects with large overjets (≥ 3.5 mm) had more muscle impairment (38.3%).

Table 27 is a distribution of TMJ pain across patient attributes. The largest subgroup of Angle Class II subjects

with the same patient attributes had the largest number of subjects with posterior TMJ pain, a severe impairment.

Interestingly, the other two groups of severe impairment were found in subjects with deep overbites as well, in contrast however, they had overjets of 3.0 mm or

Table 27

Frequency and Percentage Distribution of the Pretreatment TMJ Pain (D) Across Pretreatment Attributes

Angle Class	Over bite	Over jet	Cross bite	N	TMJ Pain Percent (N)		
					0	1	5
I	≤3.0	≤3.0	No	8	100.0(8)	0	0
I	≤3.0	≤3.0	Yes	5	100.0(5)	0	0
I	≤3.0	≥3.5	No	6	100.0(6)	0	0
I	≤3.0	≥3.5	Yes	0	0	0	0
I	≥3.5	≤3.0	No	12	75.0(9)	8.3(1)	16.7(2)
I	≥3.5	≤3.0	Yes	2	100.0(2)	0	0
I	≥3.5	≥3.5	No	2	100.0(2)	0	0
I	≥3.5	≥3.5	Yes	0	0	0	0
II	≤3.0	≤3.0	No	10	100.0(10)	0	0
II	≤3.0	≤3.0	Yes	2	50.0(1)	50.0(1)	0
II	≤3.0	≥3.5	No	4	100.0(4)	0	0
II	≤3.0	≥3.5	Yes	2	100.0(2)	0	0
II	≥3.5	≤3.0	No	13	84.6(11)	0	15.4(2)
II	≥3.5	≤3.0	Yes	4	100.0(4)	0	0
II	≥3.5	≥3.5	No	18	72.2(13)	11.1(2)	16.7(3)
II	≥3.5	≥3.5	Yes	2	100.0(2)	0	0
III	≤3.0	≤3.0	No	2	100.0(2)	0	0
III	≤3.0	≤3.0	Yes	2	100.0(2)	0	0
III	≤3.0	≥3.5	No	0	0	0	0
III	≤3.0	≥3.5	Yes	0	0	0	0
III	≥3.5	≤3.0	No	0	0	0	0
III	≥3.5	≤3.0	Yes	1	100.0(1)	0	0
III	≥3.5	≥3.5	No	0	0	0	0
III	≥3.5	≥3.5	Yes	0	0	0	0
Total				95	84	4	7

less. These subjects had the conditions for a trapped mandible and supports the clinical viewpoint of a greater incidence of TMJ pain in these types of subjects. In contrast, if the subjects had deep overbites, small overjets, but crossbites, then no TMJ pain occurred (N=7).

TMJ pain was not influenced by crossbites, in general, or by overjet and Angle Classification. Subjects with overbites less than or equal to 3 mm had almost no TMJ pain (97.6%) compared to subjects with 3.5 mm or greater overbite (81.5%). In addition, 13.0% of the deep bites had posterior TMJ pain.

Table 28 is the distribution of pain with mandibular movement across patient attributes. Of the 18 subjects comprising the largest subgroup of Angle Class II subjects, 88.9% had no pain with mandibular movement. This subgroup also had one of just two subjects with pain on more than one mandibular movement, a severe impairment. In the other large subgroup of subjects, the Angle Class II subjects with deep bites, small overjets, and no crossbites, 84.6% of the subjects were without mandibular movement pain. In the subgroup of Angle Class I subjects with deep overbites, small overjets, and no crossbites, 100% of the group's subjects were without any mandibular movement pain pretreatment.

There was a trend of increasing percentage of impairment noted from Angle Class I to Class III subjects. No pain on mandibular movement was found in all 35 Angle Class I patients, and in 89.1% of the Angle Class II

Table 28

Frequency and Percentage Distribution of the Pretreatment Pain on Movement of the Mandible (E) Across Pretreatment Attributes

Angle Class	Over bite	Over jet	Cross bite	N	<u>Pain on Mandibular Movement</u> Percent (N)		
					0	1	5
I	≤3.0	≤3.0	No	8	100.0(8)	0	0
I	≤3.0	≤3.0	Yes	5	100.0(5)	0	0
I	≤3.0	≥3.5	No	6	100.0(6)	0	0
I	≤3.0	≥3.5	Yes	0	0	0	0
I	≥3.5	≤3.0	No	12	100.0(12)	0	0
I	≥3.5	≤3.0	Yes	2	100.0(2)	0	0
I	≥3.5	≥3.5	No	2	100.0(2)	0	0
I	≥3.5	≥3.5	Yes	0	0	0	0
II	≤3.0	≤3.0	No	10	90.0(9)	10.0(1)	0
II	≤3.0	≤3.0	Yes	2	100.0(2)	0	0
II	≤3.0	≥3.5	No	4	100.0(4)	0	0
II	≤3.0	≥3.5	Yes	2	100.0(2)	0	0
II	≥3.5	≤3.0	No	13	84.6(11)	15.4(2)	0
II	≥3.5	≤3.0	Yes	4	75.0(3)	25.0(1)	0
II	≥3.5	≥3.5	No	18	88.9(16)	5.6(1)	5.6(1)
II	≥3.5	≥3.5	Yes	2	100.0(2)	0	0
III	≤3.0	≤3.0	No	2	50.0(1)	0	50.0(1)
III	≤3.0	≤3.0	Yes	2	100.0(2)	0	0
III	≤3.0	≥3.5	No	0	0	0	0
III	≤3.0	≥3.5	Yes	0	0	0	0
III	≥3.5	≤3.0	No	0	0	0	0
III	≥3.5	≤3.0	Yes	1	100.0(1)	0	0
III	≥3.5	≥3.5	No	0	0	0	0
III	≥3.5	≥3.5	Yes	0	0	0	0
Total				95	88	5	2

subjects, but only 80% of the Angle Class III subjects were pain free. Crossbite, overbite, and overjet did not appear to have any influence on pain with mandibular movement.

This lack of baseline homogeneity across these five clinical signs made the determination of which patient attributes were possible risk factors for TMD impossible. In other words, base line conditions can influence treatment and do influence baseline symptoms, but because treatment is not homogeneous across baseline conditions, that is, patient attributes, treatment is not homogeneous across baseline signs.

In addition, the limited groups, inadequate sample size, and the fact that some attributes helped some of the clinical signs while making other signs worse, lends support to the evaluation of the clinical signs of dysfunction individually rather than as a total indices score. It is evident that the pretreatment conditions are associated with, or influence the severity of the pretreatment signs. Patient pretreatment conditions, patient attributes and the pretreatment signs and symptoms, can be expected to affect the final signs and symptoms at the end of orthodontic treatment.

Out of 95 patients, only a group of eight subjects had the same pretreatment patient attributes, and only seven had the same treatment modalities. Six of the eight were

female. Only five of these subjects had no impairment in any of the five clinical signs evaluated, one had a severe impairment (TMJ pain), one had slight muscle impairment, and the last had slight TMJ (pain) and mandibular movement pain.

Evaluation of the Anamnestic Index

A number of comparisons are possible related to the frequency of symptoms (APPENDIX A, pg. 179) reported, summarized in Table 29, and pretreatment signs reported on Tables 8-9, 15-19.

The first two questions assist in the description of the present sample. Only five of 95 had previous orthodontic treatment, and only one person reported treatment for TMJ pain or dysfunction. Interestingly, question one regarding orthodontic treatment appeared to cause increasing confusion even after active orthodontic treatment was discontinued.

A large number of subjects, 28.4%, had awareness of TMJ sounds (question 3) pretreatment, and this gradually decreased, by the subject's assessment, to 18.3% after completion of orthodontic treatment. This compares to 38.9% of the patients who demonstrated sounds and/or mandibular deviation upon opening at the pretreatment examination.

Muscle stiffness upon awakening (question 4) (possibly due to nocturnal bruxism) was reported by 8.4% of the subjects pretreatment. This increased slightly during

Table 29

Percentage Distribution of Positive Responses in the Anamnestic Dysfunction Index in the Sample From Pretreatment to Posttreatment Interval II

TIME	Anamnestic Dysfunction Index Questions									
	1	2	3	4	5	6	7	8	9	10
PRETREATMENT										
N= 95	5.3	1.1	28.4	8.4	11.6	13.7	7.4	3.2	8.4	17.9
DURING TREATMENT										
N= 95	30.5	1.1	26.3	9.5	8.4	11.6	6.3	5.3	6.3	16.8
POSTTREATMENT I										
N= 94	38.3	0	18.3	7.4	8.5	5.3	3.2	3.2	7.4	23.4
POSTTREATMENT II										
N= 60	55.0	1.7	20.0	6.7	10.0	11.7	5.0	8.3	11.7	20.0

treatment, but then decreased after orthodontic treatment ended and continued to decrease throughout the posttreatment, or retention period.

Initial subjective masticatory muscle pain (question 5) was reported by only 11.6% of the subjects pretreatment, although 31.6% of the subjects (Table 15) had at least one painful muscle upon clinical examination. The symptom decreased only slightly by the end of orthodontic treatment to 8.5%, and then increased during retention to the pretreatment level. The 11.6% corresponds well with the

clinical results in which 8.4% of the patients had four or more muscle sites tender to palpation pretreatment.

Subjective limited maximum opening (question 6) was reported by 13.7% pretreatment, which does not correspond to the clinical exam which found no subject unable to open at least 40 mm pretreatment. By the end of orthodontic treatment 8.4% felt they improved, however, into retention, 11.7% of the subjects reported limited maximum opening.

Subjective reports of open locks (question 7) decreased through the course of treatment from 7.4% to 3.2%, however during retention this increased to 5% of the subjects. This low value underscores what was previously stated in the literature review, that many signs and symptoms of TMD are transient, and this causes many of the difficulties from a cross-sectional study design. In this sample, locking was only found in one person pretreatment.

Subjective pain upon mandibular movement (question 8) increased slightly during orthodontic treatment, and returned to pretreatment levels postorthodontic treatment. However, by retention, 8.3% of the subjects reported pain with mandibular movement. Clinically, only 7.4% (Table 19) had pain with mandibular movements pretreatment. Possibly the increase in retention is due to the increase in trauma reported in question 10.

Subjective TMJ or muscle pain (question 9) gradually decreased throughout treatment, but increased beyond pretreatment levels during orthodontic retention.

Trauma (question 10) to the jaws was a relatively common occurrence in the subjects pretreatment, 17.9%, and in general increased throughout orthodontic treatment.

Evaluation of the Influence of Orthodontic
Treatment on the Components of the
Clinical Dysfunction Index

Impaired Range of Movement/Mobility Index

Mandibular mobility during orthodontic treatment, improved for 16 of 20 subjects (80.0%), whereas only 18.1% developed a worse condition than pretreatment (Table 30). Of the entire sample, only 65% remained the same once into treatment. In otherwords, patients who start out with a mobility impairment pretreatment have a significant (80%) chance, or four to one odds, of getting better during orthodontic treatment.

Table 35 shows what happens to those subjects at the end of treatment, 0 to 13 months after active fixed appliances are removed. Most of the entire sample, 54.74% had no impairment of mandibular mobility pretreatment, during treatment, and into posttreatment. All 18 of the subjects of the Angle Class II subgroup from Tables 18-21 were part of this asymptomatic group. A small number, ten (10.5%) of the entire sample did have more mandibular

mobility impairment posttreatment, including the 6.32% who had no impairment at the beginning of treatment, or during treatment. For the entire sample, 21 (22.1%) returned to the pretreatment baseline level of impairment. The one person with the severe mobility impairment pretreatment (Table 9), had some resolution during treatment which maintained itself to posttreatment.

Initially asymptomatic subjects who developed some level of mobility impairment during treatment had 14 to 3 odds, or 82%, chance of returning to an asymptomatic state by the end of active treatment.

Table 35 shows that of the 15 patients who had mild mobility impairment pretreatment which resolved during treatment, 66.67% of them maintained this asymptomatic mandibular mobility through posttreatment, and the other 33.33% returned to their mild pretreatment level. Four subjects had slight mobility impairment which did not resolve during treatment. Of these four, one resolved by posttreatment, two stayed the same, and one became worse.

Impaired TMJ Function

Impaired TMJ function was the most common clinical finding pretreatment (Table 16). Table 31 shows that of the 57 subjects without any pretreatment TMJ function impairment, 12 (21%) developed an impairment during treatment. However, when considering the 94 subjects who

could have become impaired or gotten worse during treatment, only 12.8% did. This can be compared to 87.4% of the sample who stayed the same or improved during treatment.

Patients who began with an impairment, had only a 42% (16/38) chance of improving from pretreatment to approximately one year into treatment. However, the odds are 8 to 11 of improving over staying the same.

Table 36 shows what happened to the sample into posttreatment with regards to impaired TMJ function. Almost 19% (18 subjects) had a worse impairment posttreatment than pretreatment. Just 35.8% of the sample had no TMJ impairment from pretreatment through the completion of orthodontic treatment. These two figures suggest the transient and fluctuant nature of TMJ sounds and mandibular deviations which occurred in the remainder of the sample.

Of the 12 people who developed an impairment in TMJ function during treatment (from Table 31), six improved by the end of treatment with five (41.7%) of the six having complete resolution of their problems. Of this same group of twelve, five subjects became impaired during treatment, and remained at that level after treatment. Only one person actually had a progression through treatment to a severe impairment by the end of treatment. Therefore, a patient who develops a TMJ impairment during treatment only has a 50% chance of that improving by the end of treatment, and

possibly less if some of the 11 subjects who had slight impairment in posttreatment actually developed the impairment from the unevaluated interval during treatment to deband.

Of the 38 subjects beginning treatment with a slight impairment, 15 (40.5%), resolved their impairment by the end of treatment, and 22 (59.5%) stayed the same after orthodontic treatment. It appears from Table 34, that if the subjects experienced the improvement in the first year of treatment, over half (56.3%) would maintain that improvement until the end of treatment. Subjects who begin with a slight TMJ impairment which does not resolve during treatment, have only a 28.6% chance of that impairment resolving. Or, they have two to five odds of that impairment resolving versus staying the same. The negative effect is that 18.9% of the subjects developed TMJ function impairment or more impairment at the end of orthodontic treatment, however, only one of which was severe. Only one person started with locking, which improved by the end of treatment.

Six of the Angle Class II group from Tables 21-28 were asymptomatic from the beginning through the end of treatment. Two of the subjects of this subgroup, began treatment with slight impairment which resolved during treatment and stayed asymptomatic posttreatment.

Muscle Pain

Almost 59% of the subjects had no painful muscle sites pretreatment or during treatment (Table 32). About 67% of the sample did not change their level of muscle impairment during treatment. Only 9 (9.7%) subjects who could only have developed impairments during treatment did, compared to 22 (73.3%) of those who improved. Subjects with pretreatment muscle pain had 11:4 odds of having decreased number of painful masticatory muscles after one year of orthodontic treatment.

Table 37 shows what happened to the sample's muscle pain at the end of treatment. Almost 52% of the sample had no painful muscles from the beginning until the end of treatment. Only 11 subjects were worse after orthodontic treatment than they were pretreatment with respect to the number of painful muscle sites. Of the 56 subjects who had no painful muscles either pretreatment or during treatment (Table 32), seven developed 1 to 3 painful masticatory muscles by the end of treatment. This is in contrast to the nine asymptomatic pretreatment subjects who developed painful masticatory muscles during treatment, all of whom improved by the end of treatment. Of the nine, seven became completely asymptomatic by the end of treatment. All seven of the subjects had developed one to three painful muscles during treatment.

Some subjects beginning treatment with impairment (N=30) had resolution of those painful muscles during the first year of treatment which lasted throughout the remaining treatment (46.7%). Additionally, subjects continued to improve from during treatment to the end of treatment in approximately the same proportions, so that by the end of treatment, 83.3% of those with pretreatment impairment were improved by the end of treatment. In otherwords, lack of improvement during the first year of treatment was not an indication that the chances of improvement would be less during the remaining treatment. A small number of subjects 22.7% (N= 5) who started with slight muscle impairment stayed the same or became worse by the end of treatment. Subjects who had slight muscle problems pretreatment had two to one odds of having no painful muscle sites at the end of treatment.

Eight subjects who started out with severe muscle impairment pretreatment, all improved by the end of treatment. Of this severe group, four became asymptomatic by the end of treatment. Subjects with severe muscle impairment pretreatment, only had a 50% chance of becoming asymptomatic by the end of treatment in contrast to 86% for pretreatment asymptomatic subjects, and 77% for slightly impaired subjects pretreatment.

The negative effect is that 11.6% of the subjects developed painful muscles or more muscles that were painful at the end of orthodontic treatment, only two of which were severe.

Six of the Angle Class II subgroup from Tables 21-24 were without muscle pain pretreatment throughout posttreatment. One individual started with slight muscle impairment which resolved by posttreatment, while another subject developed slight muscle impairment by the end of treatment.

TMJ Pain

TMJ pain was a relatively infrequent finding. Table 33 shows that almost 79% of the sample did not have TMJ pain pretreatment or during treatment. Of the 84 subjects who had no TMJ pain pretreatment, two developed lateral palpation pain and seven developed posterior pain during treatment. Eleven subjects had TMJ pain pretreatment, of which, nine (81.8%) completely resolved during treatment, and two remained the same. In otherwords, subjects have nine to two odds of having complete resolution of TMJ pain within the first year of active orthodontic treatment.

Pretreatment through posttreatment, 72.6% of the sample was asymptomatic for TMJ pain. Table 38 shows that only six (8.0%) of the sample became symptomatic by the completion of orthodontic treatment, compared to 20 (21.1%) of the

subjects who started with TMJ pain or developed it during treatment, and were all asymptomatic by the completion of treatment. All nine of the pretreatment asymptomatic individuals who developed TMJ pain by the first year into treatment returned to their asymptomatic pretreatment level.

Only 6.3% of the subjects developed TMJ pain laterally or posteriorly by the end of orthodontic treatment, two of which were severe. Both of these subjects were without pretreatment impairment.

Six of the eight Angle Class II subgroup from Tables 21-28 were asymptomatic from the beginning of treatment through the end. Two subjects started with slight and severe impairment which resolved during treatment and remained asymptomatic posttreatment.

Pain on Mandibular Movement

Finally, Table 36 shows that of the 88 subjects who started treatment without any pain on mandibular movements, five (4.8%) developed pain in one movement by the end of the first year of treatment. Four (57.1%) of the seven subjects who started with pain on mandibular movement had the pain resolve or improve during treatment. Only one subject with pain in one movement developed a worse condition during treatment. Subjects with mandibular movement pain had five to two odds of having some or total resolution of that pain during orthodontic treatment.

Table 39 shows that by the completion of treatment, only five (5.3%) of the entire sample had more mandibular movements that were painful than pretreatment. Pretreatment through posttreatment, 83.2% of the subjects were asymptomatic in terms of mandibular movement pain. This is compared to the five out of seven (71.4%) who had complete resolution of their pretreatment problem. Of the two people who had a problem pretreatment and became worse during treatment, one returned to the pretreatment level, and one remained the same. Of the five people who developed a slight problem during treatment, four (80%) had complete resolution by the end of treatment, and one remained the same. In otherwords, patients who develop pain with mandibular movement pretreatment have four to one odds of having that sign resolve by the end of treatment. Of the five subjects with pain in one movement pretreatment, three had complete resolution by the end of treatment, and the other two remained the same.

Seven of the Angle Class II subgroup from Tables 22-29 were asymptomatic from the beginning throughout treatment. One subject had slight impairment pretreatment which resolved during treatment and remained asymptomatic through posttreatment.

Finally, APPENDIX B and D data show that it is not the same individuals with improvement in mandibular mobility

from pretreatment to posttreatment who had improvement in other impaired signs as well. In otherwords, the improvement and impairment of the five signs appears to have occurred independent of each other. A possible reason for this is that change is largely dependent upon the individual's ability to experience improvement.

Of the 95 subjects in the sample, 65 (68.4%) experienced change in one or more of the clinical dysfunction signs from pretreatment to posttreatment. Most (N=29) experienced only improvement; 17 in only one sign, ten in two signs, and two in three signs. Almost 28% (N=18), developed an impairment or experienced a worsening of their pretreatment impairment. Only four of these subjects experienced this decline in two of the five possible signs, and no subject worsened in three or more.

Finally, the remaining 18 subjects experienced a "mixed" result pretreatment to posttreatment, that is, they improved in one or more sign, but also developed or deteriorated in one or more other sign. The clinical signs while related do not appear to be absolutely dependent upon each other.

Table 30

Transition From Pretreatment to
During Treatment for Impaired Range
of Movement/Mobility Index (A)

Pretreatment	During Treatment			Total
	0 None	1 Slight	5 Severe	
0 None	58 * 77.33	16 21.33	1 1.33	75 100.00
1 Slight	15 78.95	4 21.05	0 0.00	19 100.00
5 Severe	0 0.00	1 100.00	0 0.00	1 100.00
Total	73	21	1	95 100.00

* Frequency
Row Percent

Table 31

Transition From Pretreatment to
During Treatment for Impaired TMJ Function (B)

Pretreatment	During Treatment			Total
	None	1 Sounds/ Deviation	5 Locking	
0 None	45 * 78.95	11 19.30	1 1.75	57 100.00
1 Sounds/ Deviation	16 43.24	21 56.76	0 0.00	37 100.00
5 Locking	0 0.00	0 0.00	1 100.00	1 100.00
Total	61	32	2	95 100.00

* Frequency
Row Percent

Table 32

Transition From Pretreatment
to During Treatment for Muscle Pain (C)

Pretreatment	During Treatment			Total
	0 None	1 1-3 Sites	5 4 or More	
0 None	56 * 86.15	7 10.77	2 3.08	65 100.00
1 1-3 Sites	16 72.73	6 27.27	0 0.00	22 100.00
5 4 or More	2 25.00	4 50.00	2 25.00	8 100.00
Total	74	32	2	95 100.00

* Frequency
Row Percent

Table 33

Transition From Pretreatment
to During Treatment for TMJ Pain (D)

Pretreatment	During Treatment			Total
	0 None	1 Lateral	5 Posterior	
0 None	75 * 89.29	2 2.38	7 8.33	84 100.00
1 Lateral	4 100.00	0 0.00	0 0.00	4 100.00
5 Posterior	5 71.43	0 0.00	2 28.57	7 100.00
Total	84	2	9	95 100.00

* Frequency
Row Percent

Table 34

Transition From Pretreatment to During Treatment
for Pain on Movement of the Mandible (E)

Pretreatment	During Treatment			Total
	0 None	1 One Movement	5 Two or More	
0 None	83 * 94.32	5 5.68	0 0.00	88 100.00
1 One Movement	3 60.00	1 20.00	1 20.00	5 100.00
5 Two or More	1 50.00	1 50.00	0 0.00	2 100.00
Total	87	7	1	95 100.00

* Frequency
Row Percent

Table 35

Transitions From Pretreatment
and During Treatment to Posttreatment
for Impaired Range of Movement/Mobility Index (A)

Pretreatment and During Treatment	Post Treatment			Total
	0 None	1 Slight	5 Severe	
00 None\None	52 * 89.66	6 10.34	0 0.00	58 100.00
01 None/Slight	13 81.25	3 18.75	0 0.00	16 100.00
05 None/Severe	1 100.00	0 0.00	0 0.00	1 100.00
10 Slight/None	10 66.67	5 33.33	0 0.00	15 100.00
11 Slight/Slight	1 25.00	2 50.00	1 25.00	4 100.00
15 Slight/Severe	0 0.00	0 0.00	0 0.00	0 0.00
50 Severe/None	0 0.00	0 0.00	0 0.00	0 0.00
51 Severe/Slight	0 0.00	1 100.00	0 0.00	1 100.00
55 Severe/Severe	0 0.00	0 0.00	0 0.00	0 0.00
Total	77	17	1	95 100.00

* Frequency
Row Percent

Table 36

Transitions From Pretreatment
and During Treatment to Posttreatment
for Impaired TMJ Function (B)

Pretreatment and During Treatment	Post Treatment			Total
	0 None	1 Sounds/ Deviations	5 Locking	
00 None/None	34 * 75.56	11 24.44	0 0.00	45 100.00
01 None/ Sounds Deviation	5 45.45	5 45.45	1 9.09	11 100.00
05 None/ Locking	0 0.00	1 100.00	0 0.00	1 100.00
10 Sounds Deviation /None	9 56.25	7 43.75	0 0.00	16 100.00
11 Sounds Deviation /Sounds Deviation	6 28.57	15 71.43	0 0.00	21 100.00
15 Sounds Deviation /Locking	0 0.00	0 0.00	0 0.00	0 0.00
50 Locking/ None	0 0.00	0 0.00	0 0.00	0 0.00
51 Locking/ Sounds & Deviation	0 0.00	0 0.00	0 0.00	0 0.00
55 Locking/ Locking	0 0.00	1 100.00	0 0.00	1 100.00
Total	54	40	1	95 100.00

* Frequency
Row Percent

Table 37

Transitions From Pretreatment
and During Treatment to Posttreatment
for Muscle Pain (C)

Pretreatment and During Treatment	Post Treatment			Total
	0 None	1 1-3 Sites	5 4 or More	
00 None/None	49 * 87.50	7 12.50	0 0.00	56 100.00
01 None/ 1-3 Sites	7 100.00	0 0.00	0 0.00	7 100.00
05 None/ 4 or More	0 0.00	2 100.00	0 0.00	2 100.00
10 1-3 Sites/ None	13 81.25	2 12.50	1 6.25	16 100.00
11 1-3 Sites/ 1-3 Sites	4 66.67	1 16.67	1 16.67	6 100.00
15 1-3 Sites 4 or More	0 0.00	0 0.00	0 0.00	0 0.00
50 4 or more/ None	1 50.00	1 100.00	0 0.00	2 100.00
51 4 or More/ 1-3 Sites	1 25.00	3 75.00	0 0.00	4 100.00
55 4 or More/ 4 or More	2 100.00	0 0.00	0 0.00	2 100.00
Total	77	16	2	95 100

* Frequency
Row Percent

Table 38

Transitions From Pretreatment
and During Treatment to Posttreatment
for TMJ Pain (D)

Pretreatment and During Treatment	Post Treatment			Total
	0 None	1 Lateral	5 Posterior	
00 None/None	69 * 92.00	4 5.33	2 2.67	75 100.00
01 None/ Lateral	2 100.00	0 0.00	0 0.00	2 100.00
05 None/ Posterior	7 100.00	0 0.00	0 0.00	7 100.00
10 Lateral/ None	4 100.00	0 0.00	0 0.00	4 100.00
11 Lateral/ Lateral	0 0.00	0 0.00	0 0.00	0 0.00
15 Lateral/ Posterior	0 0.00	0 0.00	0 0.00	0 0.00
50 Posterior/ None	5 5.26 100.00	0 0.00 0.00	0 0.00 0.00	5 100.00
51 Posterior Lateral/	0 0.00 0.00	0 0.00 0.00	0 0.00 0.00	0 0.00
55 Posterior Posterior	2 2.11 100.00	0 0.00 0.00	0 0.00 0.00	2 100.00
Total	89	4	2	95 100.00

* Frequency
Row Percent

Table 39

Transitions From Pretreatment
and During Treatment to Posttreatment
for Pain on Movement of the Mandible (E)

Pretreatment and During Treatment	Post Treatment			Total
	0 None	1 One Movement	5 Two or More	
00 None/None	79 * 95.18	3 3.16	1 1.20	83 100.00
01 None/ One Movement	4 80.00	1 20.00	0 0.00	5 100.00
05 None/Two or More	0 0.00	0 0.00	0 0.00	0 0.00
10 One Movement/None	3 100.00	0 0.00	0 0.00	3 100.00
11 One Movement/ One Movement	0 0.00	1 100.00	0 0.00	1 100.00
15 One Movement/ Two or More	0 0.00	1 100.00	0 0.00	1 100.00
50 Two or More/ None	1 100.00	0 0.00	0 0.00	1 100.00
51 Two or More/One Movement	1 100.00	0 0.00	0 0.00	1 100.00
55 Two or More/ Two or More	0 0.00	0 0.00	0 0.00	0 0.00
Total	88	6	1	95 100.00

* Frequency
Row Percent

DISCUSSION

Reliability

A standardized method of the measurement of TMD signs and symptoms allows valid comparisons as to the efficacy of orthodontic treatment, response of the signs and symptoms to orthodontic treatment, and the determination factors which affect the sign and symptoms. This type of methodology is central to longitudinal study such as the Iowa TMD Study, as is the need for reliability and validity of the measurement. Reliable measurement of TMD signs and symptoms longitudinally would allow cause and effect factors to be identified as well as the evaluation of the effect of orthodontic treatment.

"Because the validity of any measurement can only be equal or less than its reliability, epidemiologic data gathered through clinical examiners with unknown reliability is of unknown validity and hence of questionable use in population-based studies of diagnosis" (Dworkin et al., 1990a). It is this premise which makes the calibration process so critical. Results from Table 3 indicate that two examiners in the Iowa TMD Study are able to measure the signs and symptoms reliably and accurately from one year to

the next compared to each other. However, we are not able to determine if measurement collection has maintained its same consistency and accuracy for intra-examiner measurements or from the initial examiners to the present examiners. The only way to do this would be to have recalibrated each year on a manikin which was adjustable for the signs and symptoms measured.

Recalibration on the same group of subjects each year to determine if reliability of the measurement technique had diverged over time would not be a alternative. Patient variability of their signs and symptoms over time would be a major limitation. Examiners would not necessarily examine the same signs year to year or even day to day because of their fluctuant nature. Likewise, control groups for comparison in a longitudinal TMD study would be of limited benefit for the same reasons, and only increase error.

It is interesting to note that the greatest variation between the present examiners occurred in measurement of overbite which involved a ruler. The apparent reason for this discrepancy was that examiners may not have averaged the overbite if it was not the same for both right and left central incisors, but instead recorded the largest value. The other parametric variables which showed lack of agreement involved measurements of unassisted lateral movements. The reason for this is patients may actually

have moved downward and forward which would have given a shorter measurement than actually possible.

Finally, as expected, TMJ function which involves the measurement of TMJ sounds and/or deviations was the most variable nonparametric sign evaluated, in spite of the weighted index. It is possible for this sign to be subclinical initially and express its self at subsequent examinations. Many subjects have intermittent joint sounds which may or may not be correlated to episodes of impairment and pain. The extreme variability of this particular sign makes its clinical usefulness as a diagnostic or predictive sign limited.

These findings are similar to those reported by Dworkin and others (1990), however greater agreement between those examiners in parametric variables was probably due to the shorter retraining periods and more thorough definitions as to how and where the examination measurements were to be taken.

Although the Helkimo Indices used in this study were not designed to be diagnostic for TMD, by dividing them into their various components it was hoped it would be possible to identify a number of risk factors, both patient attributes and orthodontic treatment modalities, which precipitated or alleviated the measured TMD signs and symptoms. The small sample size has not yet permitted the

longitudinal study to determine these factors or their cause/effect relationships.

This study evaluated the longitudinal effects of orthodontic treatment on signs and symptoms as measured by the Helkimo Indices. It is appropriate to use the Helkimo Indices for this type of study without diagnosing the type of TMD present because these are not TMD patients. Only one of the patients specifically reported treatment for TMD prior to the orthodontic treatment. Many of the measured signs are characteristic of many possible variations of TMD, and since it was not clear at this early stage which form of TMD, if any, the subjects would develop, it was reasonable to follow the progression of the five general signs. The study used the Helkimo Indices to facilitate determination of changes in the signs and symptoms while undergoing orthodontic treatment. The argument made by van der Weele and Dibbets (1987) that the Helkimo Indices do not properly identify patients with mild TMD, that is, they lack the specificity to determine the TMD patient population, did not detract from its use in this study as a method to determine frequency and severity of signs.

Some of the measured signs and symptoms, for example sounds, have not proven to be as clinically relevant or important for treatment or diagnosis of TMD as they were thought when the Helkimo Indices were introduced in 1972.

This is an expected dilemma with medical/epidemiological progress, and a longitudinal study such as the Iowa TMD study.

In spite of these limitations, the Helkimo Indices in the context of this study appear to have been a consistent, reliable method to determine changes which occurred in the signs and symptoms of TMD. Its sensitivity, that is, how often the index detected signs and symptoms versus how many occurred in the sample was really contingent on examination technique, measurement, and patient variability. Previous reliability studies suggested that the most sensitive method to collect sound measurements was a stethoscope, although palpation is considered equally valid (Dworkin *et al.*, 1990). This area showed the consistent variation in each year of calibration, see Table 3. Muscle palpation was performed by digital palpation and not an algometer. This was not an area of disagreement between the present two examiners possibly because the score was indexed and because the reliability sample was basically asymptomatic. Finally, mobility measurements were made with the use of a plastic ruler which should have facilitated the least variability. Overbite and overjet had the greatest variability due to differences in adjusting for varied amounts in the same patient.

Overall, the examiners calibrated for this study had greater agreement, and more reliability than results reported by Dworkin and others (1990b). The most likely reason for this is the common educational, clinical and skill level of the two examiners. Dworkin reported on the results of dental hygienists who were trained in the examination technique. Also, the reliability sample, like the study sample, consisted of basically healthy subjects who had no impairment or slight impairment of the signs of the clinical dysfunction index. This high frequency of "zero" scores also helped the reliability between examiners.

In addition to examiner error is the error due to the variability of the signs themselves. TMD signs and symptoms may change in character and have active and quiescent periods which act in cyclical patterns lasting years. Because the factors which cause arrest or remission are unknown, caution must be used for evaluation of successful alleviation of the signs and symptoms of TMD. Past cross sectional studies have given the appearance that TMD is progressive in nature, however longitudinal epidemiologic studies suggest that many signs of TMD are self-limiting. Without this understanding, evaluation of sounds, mobility, pain, and other specific signs and symptoms can lead to a misunderstanding of their response to orthodontic treatment. Simple methods, other than patient's subjective pain, are

not available to the clinician to determine the presence of active disease.

The sample was selected based on several requirements stated in the methods and materials. One of the requirements was that the posttreatment Helkimo score was within 13 months after deband. The sample may have been biased in that subjects with severe problems may have been less likely to return for retention check appointments and were therefore excluded from the study based on the severity of their symptoms. No evaluation was performed to evaluate the homogeneity of the collection of the treatment scores. There are 10 to 20 subjects in the entire study thus far who do not have posttreatment Helkimo scores or whose posttreatment scores are over 13 months after completion of their fixed orthodontic treatment.

Frequency of Signs and Symptoms

In general, the frequency of symptoms reported by the subjects in this study is similar to the frequency reported by other pre- and posttreatment orthodontic patients, but lower than reported by the general population.

Of the present study's subjects, 66 (69.5%) reported having none of the questioned symptoms. This frequency compares well with the 80% Wannman and Agerberg (1986) found in 17-year-olds, and the 58% Locker and Slade (1988) found in their sample of adults 18 years of age and older.

Compared to other orthodontic samples reported in the literature review, this frequency of symptom free individuals falls between the 57% reported by Janson and Hasund (1981), and the 73% reported by Larsson and Ronnerman (1981). It is not in good agreement with others (Helkimo, 1974; Dahl et al., 1988; Pullinger et al., 1988b; Schiffman et al., 1990). Of the 66 subjects, only 15% reported a history of trauma. Some of these differences can be explained by the differently worded questions, format, or broader age groups and different levels of oral health.

The frequency of TMJ sounds pretreatment, 28.4%, is within the 21 to 39% reported by other studies (Table 1). It is not in agreement with the 13% found in 17-year-old subjects (Wannman et al., 1986) or with Salonen and others (1990) who used a question with a multiple choice response.

The frequency of TMJ pain symptoms is not readily comparable due to the combined form of the question, that is, muscle pain as well as TMJ pain. This sample of subjects reported TMJ and muscle pain in 8.4% of the subjects pretreatment, and 11.6% reported muscle pain specifically.

The sample's frequency of pretreatment symptoms of pain with mandibular movement does agree with the frequency found by Wannman and Agerberg (1986) and Agerberg and Inkapool (1990). It is not in agreement with higher frequencies

reported by Helkimo (1974) and Salonen and others (1990) possibly due to the broader age ranges.

Finally, the reported symptom of decreased opening, 13.7%, is close only to the cross sectional study of Reider and others (1983) who found 14.1% felt they had limited opening. This frequency is about double of what is reported by the other six studies, two of which found extremely low frequencies. A possible reason for the high frequency reported by this sample is because their malocclusions may have led them to believe they could not open as wide as their peers.

The frequency of signs in the pretreatment sample closely agrees with the majority of the frequencies reported in the literature reviewed. The comparison of both the symptoms and the signs with the literature review indicates that the sample was representative of the general population and of typical orthodontic patients.

In this sample, 38.9% of the subjects pretreatment were without any dysfunction in the five areas evaluated in the clinical dysfunction index. This frequency of D_10 is in agreement with five of the epidemiologic studies reported and two of the orthodontic studies which reported postorthodontic treatment results.

The most frequent pretreatment sign found was TMJ sounds and or deviation which occurred in 40% of the sample.

This was in close agreement with three of the epidemiologic studies. The frequency of sounds in the studies in the literature review for less than 30-year-old subjects ranged from 17% to 53%. Some of the differences are due to studies separating the popping sounds from the crepitus sounds. In general, this study's sample did not appear to be vastly different than the general population with regards to TMJ sounds. Therefore, we would expect the natural course of TMJ sounds to follow a similar time line as reported by Rieder and others (1983), Gross and Gale (1983), Helkimo (1974), and Salonen and others (1990). That is, that TMJ sounds tend to increase in frequency from early childhood to young adulthood, then slowly increase to a peak frequency in the 30 to 50 year old range and then remain at that same frequency (30 to 71%) through the older age groups.

Over the two years subjects were in orthodontic treatment, no large changes in frequency would be expected. Minor changes would be due to the fluctuant nature of TMJ sounds, or due to natural remission or resolution. Any changes which did occur may be attributed to the effects of the orthodontic treatment.

Deviation on mandibular opening is not distinguished as a separate sign by the Helkimo Indices, therefore it is impossible to know the actual frequency of occurrence in the present sample other than to say it can be no more than 40%,

the total pretreatment TMJ impairment. In reality, the percent of TMJ impairment due to deviation upon opening or closing is probably far less than this, perhaps as low as a third. Studies of adults found frequencies in the range of 10 to 42%. Reider and others (1983) reported that deviation upon opening of 1 mm occurred in approximately 30% of subjects 13 to 30 years of age, and gradually decreased in occurrence to about 21.1% in subjects greater than 60 years of age.

The frequency of pretreatment limited maximum opening in this sample was 0%, however, limited lateral and protrusive mobility did occur in 21.1% of the sample pretreatment. This lack of impaired maximum opening compares favorably with the low frequency of restricted maximum opening (0 to 6.5%) reported by other studies in the literature reviewed (Table 1). Limited maximum opening has the potential to be an indicator of TMD because of this low frequency. As with joint sounds, caution must prevail when determining restricted motion as a sign of TMD. Helkimo (1974) and others have hypothesized that women due to their smaller physical stature, may not be able to open as wide as men.

Impaired mandibular mobility is an infrequent finding for all adult age groups but increases in occurrence after

age 60 in the elderly (Gross and Gale, 1983; Reider et. al., 1983; Salonen et. al., 1990).

Pretreatment muscle pain, the second most frequent TMD sign, occurred in 31.6% of the subjects. This is in good agreement with the other studies reporting on a similar age group which found muscle pain occurred in 14.6 to 50% of the subjects (Table 1). The frequency of painful masticatory muscles found in this study is also in close agreement with what was found in other orthodontically treated samples.

Like many other studies, the lateral pterygoids were the most frequent site of muscle pain. This result is questionable because of the inability of being able to palpate the lateral pterygoid directly due to the anatomical limitations (Johnstone and Templeton, 1980). Most likely, the response was the medial pterygoid which had the second highest frequency of painful sites. Other studies have reported that the medial pterygoid and masseter muscles have a high frequency of pain. The high frequency of lateral pterygoid pain found may also be due to the difficulty in palpating externally. All muscle pain may be biased due to mechanical factors in palpation.

Helkimo (1974) and others found that masticatory muscle tenderness tended to increase to a range of 15 to 50% and then remain a relatively constant finding throughout all ages, but increasing slightly with age groups over 60 years

old. Of the present sample, 11.6% had pretreatment TMJ pain to palpation which is low in the range (10 to 32%) reported by other studies who used or reported findings for similar age groups. It is not as high as 45% found by Helkimo (1974) probably due to the better dental care and less "loss of posterior support" which Helkimo found associated with TMJ pain. The frequency of TMJ pain is less than what was reported on subjects who completed orthodontic treatment (Janson and Hasund, 1981; Kess et al., 1991).

TMJ pain was found to remain a relatively consistent occurrence throughout all age groups, but having the greatest occurrence in subjects 15 to 34 years of age. A reason for this peak is that individuals will probably have adapted in lifestyle, had remodeling/adaptive physical changes, or have sought treatment for the pain by their 40's.

Finally, pain with mandibular movement occurred in just 7.4% of the subjects pretreatment. This frequency fits within the range 0 to 10%, reported in the literature reviewed. It does not compare with the 24 to 38% range Helkimo (1974) found in the Lapps, again most likely due to the poor oral health of the Lapps. The frequency of mandibular movement pain also agrees with what was reported by Kess and others (1991). Cross sectional studies across large age ranges suggest that pain with mandibular movement

occurs infrequently, and for similar reasons to TMJ pain, it peaks in the 25 to 44 year old range, and gradually declines in occurrence with older age groups.

Effects of Orthodontic Treatment on TMD Signs and Symptoms

Does orthodontic treatment cause TMJ function to improve or worsen? This is the central question which can be addressed from the results of this study. Further, can orthodontic treatment be used to treat signs and symptoms of TMD disorders? Orthodontists are frequently asked these questions by potential orthodontic patients hoping to prevent TMD, or by symptomatic patients who desire treatment for relief.

The answer to the first question is that in general, orthodontics does make the five signs of TMD evaluated in this study better or leave them unchanged.

From pretreatment to posttreatment, mandibular mobility generally improved with orthodontic treatment. Of the subjects who began with mandibular mobility impairment, 60% improved while just 5% (N= 1) had more impairment after orthodontic impairment. For subjects without impairment pretreatment, just 13.3% developed slight impairment by the end of orthodontic treatment. This is not to say this improvement is necessarily the result of orthodontic treatment, however, the large differences in the individuals

who improved in an age group which has the highest occurrence of mobility impairment, suggests that orthodontics does in fact tend to improve mandibular mobility.

However, evaluation of pre- and posttreatment scores (APPENDIX B and D) shows how perplexing TMD is, that is, the same individuals with improvement in one sign did not necessarily have improvement in other impaired signs from pretreatment through posttreatment. It would be expected that the improvement or impairment of the five signs would occur in a dependent relationship of each other. For example, improvement in mandibular mobility, should be related to less muscle and TMJ pain, but this was not true for 27.7% of the subjects who changed.

With respect to impaired TMJ function from pretreatment through posttreatment, orthodontics does not appear to help TMJ function improve which agrees well with the recent University of Florida study which found joint sounds increased in frequency from 22% to 35% after two years of orthodontic treatment (Hirata et al., 1992). Of the subjects who began with TMJ impairment, just 42.1% improved although no subject finished treatment with more impairment than before orthodontic treatment. In subjects without impairment pretreatment, 31.6% had developed impairment, one of which was severe, by the end of orthodontic treatment.

This development of TMJ impairment is not necessarily the result of orthodontic treatment however because this sign is the most frequent in the general population, especially in 30- to 50-year-old subjects. The small difference in the individuals who are getting better and those who are developing TMJ impairment suggest that orthodontics does not have a significant effect on TMJ function.

Of the subjects who began with masticatory muscle pain pretreatment, 83.3% improved while just 6.7% (N=2) had increased numbers of painful muscles after orthodontic treatment. For subjects without impairment pretreatment, just 13.8% (N=9) developed one to three painful muscles which did not resolve by the end of orthodontic treatment. This is within the range of occurrence (15 to 24%) for a similar age group reported by Gross and Gale (1983), Reider and others (1983), and Salonen and others (1991). The large difference between the individuals who improve and those who worsen suggest that orthodontics does decrease the number of painful masticatory muscles.

Of the subjects who began with TMJ pain pretreatment, 100% became asymptomatic after orthodontic treatment. For subjects without pretreatment TMJ pain, just 7.1% (N= 6) developed TMJ pain which did not resolve by the end of orthodontic treatment. The large difference between the individuals who become asymptomatic compared to the small

number who developed TMJ pain suggests that orthodontics does decrease the frequency of TMJ pain.

Of the subjects who had pretreatment pain upon movement of the mandible, 71.4% became asymptomatic while just two (28.6%) remained the same, having one painful mandibular movement after orthodontic treatment. For subjects without impairment pretreatment, just 5.7% (N= 5) developed pain in one or more mandibular movements which did not resolve by the end of orthodontic treatment. This incidence of painful mandibular movements is near the level of pretreatment occurrence, and not necessarily the result of orthodontic treatment. The large number of individuals who improved suggests that orthodontics does decrease the number of painful mandibular movements.

These results underscore the importance of separation of the clinical signs from a total clinical dysfunction score to determine which specific sign is changing with orthodontic treatment. By not evaluating the Helkimo Indices as a whole score (Harrison, 1986), information about changes occurring is not lost, and changes in one sign do not offset changes occurring in another.

The results of this study, using nearly all of the same subjects reported by Kremenak and others (1992a), show that the range of patients who end orthodontic treatment at the same level of impairment as when they started ranges from

62.1% to 89.5%. Similarly, the range of subjects who had a worse impairment posttreatment than pretreatment was 5.3% to 18.9%, depending on the sign evaluated (Table 41).

Clinical use of the results in this study can help orthodontists inform patients what chances they would have of pretreatment signs and symptoms being induced, reduced, or increased with orthodontic treatment.

This of course does not explain WHY or what it is about the orthodontic treatment makes the TMD signs and symptoms improve or worsen. For this, the sample size must be large enough to compare the patients who start without impairment and develop impairment, with those who started with an impairment and improved. What do these patients have in common and in contrast to each other? What does the orthodontic treatment change to cause the effect? Is it occlusion, psyche, function? Unfortunately, the present sample is not large enough nor are the Helkimo Indices encompassing enough to address these factors.

It is interesting to note that most conservative TMD splint treatments have a success rate of 70 to 90% at the completion of treatment (Clark, 1984, 1984a). The reduction of painful signs and symptoms with appliance therapy is well documented. Although the treatment is highly predictable, the physiologic basis of the treatment response is not understood.

Mejersfo and Carlsson (1983) reported that seven years after conservative TMD therapy, over 65% of the patients had no long-term symptoms. From the same study of 154 patients, it was also concluded that most TMD patients have minimal recurrent symptoms 7 years after conservative treatment. In a 1986 study by Okeson and Hayes (1986) of 110 TMD patients, 56.4% reported no pain and 29% much less pain 2 to 8.5 years after conservative treatment. Only 14.5% of the patients were the same or worse after treatment.

Greene and Laskin (1983) added further support for the reversible treatment of MPD when they found five years after reversible therapy, 53% of the patients were asymptomatic and 37% experienced only minor residual symptoms. This was after an initial success rate of 74%.

The orthodontic treatment provided in this study approaches the same level of success. Of course orthodontic therapy provided for subjects in this study, except for possibly one person (Table 29, question 2), was never intended to treat TMD. As a side benefit, orthodontic treatment has been able to improve some of the possible signs and symptoms of TMD. Unlike other TMD modalities of therapy and contrary to most occlusal adjustment therapy, our success appears to be due to the alteration of the malocclusion. Probably, the results of subjects who only

improved was related to improved psychological well being from an esthetic result, or other unidentified changes.

Fully banded patients have the effect of being in a splint, therefore this possible "splinting effect" for the duration of treatment, on average 23 months (Table 14) in this study may also be the underlying reason for successful reduction of painful muscles and TMJ's. It is important to note the majority of the positive changes for initially symptomatic subjects occurred in the first year of treatment. On the other hand, results (Table 29) from question 4 of the anamnestic index (APPENDIX A, pg. 179) suggests that "stiff muscles" presumably from nocturnal bruxism were not decreased by this "splinting effect".

In retention, the initial success rate continued to be maintained. This is a goal of orthodontic therapy, that is, the long term resolution of the malocclusion in a state of harmony with the masticatory system. For 60 of the 95 subjects a second posttreatment score indicated that the subjects continued in a basically unchanging trend or with fluctuant change (slight impairment condition) from the first posttreatment scores. It appears as though the occlusal changes are of long term benefit to the patients.

Little data is available to show that valid distinctions (outside the range of normal) between the signs and symptoms used in the Helkimo Indices exist in nonpatient

populations and in symptomatic patients. This lack of specificity does not allow those signs (clinical measurements) and symptoms to be used as good indicators or markers of pathology.

For example the high frequency of joint sounds suggests it is not a very discriminating sign for TMD (Sutton et al., 1992). The results of the present study indicate a high frequency of change occurring throughout orthodontic treatment, which is also of unknown etiology.

Interestingly Seligman and others (1988) found deep bites with small overjets associated with patients with four or more tender masticatory muscles. The results of this study, indicate that orthodontic treatment significantly reduced the number of painful muscle sites by the end of treatment. Of course, the overbite and overjet were altered as a result of treatment. Whether this change in muscle impairment is attributable to changes in those anterior functional occlusal variables can not be shown.

Pain is probably the most discriminating factor between patients who need treatment for TMD and those who do not. Interestingly, while 7.4% to 11.6% of the sample experienced joint pain on palpation or with mandibular movement, only one subject had reported being previously treated for TMD. Herein lies the unaccountable factor, patient adaptability. Obviously many patients are able to tolerate impairment

without significant lifestyle changes. Therefore, they do not seek TMD treatment even though they have signs which warrant treatment.

In summary, the diagnostic or predictive value of following the signs and symptoms which make up the Helkimo Indices are unknown. It is quite possible they are too indiscriminate to be of any diagnostic or predictive value for TMD.

Influence of Patient Attributes On Pretreatment Signs

In this study, increasing severity of Angle Class was associated with increasing frequency of painful masticatory muscles with Angle Class III subjects having the greatest frequency of 4 or more painful muscles and Angle Class I subjects having the fewest painful muscles. These results are guarded due to the small sample size. A previous study which included 15 Angle Class III subjects found no significant relationship with muscle tenderness (Bush, 1985). Also, Angle Class was associated with increasing pain on mandibular movement, with Angle Class III patients having the greatest frequency of more than one painful mandibular movement.

Pullinger and others (1988b) found the only significant associations between muscle tenderness in four or more sites

and occlusion were: "Class II-2 was associated with more muscle tenderness than Class II-1".

This association suggests certain aspects of functional occlusion are important but, to an unknown degree (Egermark-Eriksson et al., 1983). Mounting final occlusions and initial occlusions would aid in determination of the influences of occlusal changes. The Helkimo Index alone are not sensitive or sophisticated enough to take into account the occlusal and skeletal variables or their interrelationships with one another and TMD signs and symptoms. Orthodontic treatment changes occlusion, facial appearance, self-esteem, and structural relationships, however it is uncertain which factors or what combinations are necessary to have an improvement in TMD signs.

In this study overbites of 3.5 mm or more were associated with greater numbers of painful masticatory muscles, and greater frequency of TMJ pain which agrees with the results found by De Laat and others (1986). This is a similar finding to Seligman and others (1988) who also found an association between four or more sites of muscle tenderness and deep overbites (greater than 5 mm).

Similar to Pullinger and Seligman (1991), the results here suggest a wide range of overbite measurement in the pretreatment sample, -2 to 9 mm, as well as the completely asymptomatic subjects pretreatment (38.9% of the sample),

0.5 to 6 mm. Unfortunately due to small sample size, it is not possible to make comments on the five subjects with an overbite of zero or less, nor is it possible to comment on the four subjects with overbites of 7 mm or more. Given the small differences in the two ranges, it appears as though overbite would not be a sensitive prediction factor of TMD which is in agreement with Seligman and Pullinger (1991).

From an epidemiologic basis, various morphologic components of a malocclusion may be more a potent factor in TMD development to the stomatognathic system. Even though an association between signs of TMD and overbite were found, the specific cause and effect relationships between the morphologic deviations of a malocclusion for example, deep bite, can not be determined without a longitudinal study. Given the multifactorial origin of TMD and the assumption that most patients have optimal overbites by the end of treatment, it is probable that the importance of occlusal factors will never be known.

Instead, future studies need to look for the presence of occlusal/morphological factors which "will contribute to a compromised functional occlusion and therefore, require a greater degree of adaptability from the stomatognathic system" (Lieberman et al., 1985). This set of occlusal factors when combined with particular functional

malocclusions and/or psychological states may cause a subclinical/quiescent TMD to surface and manifest itself .

Overjet was associated with the following signs, and may therefore be a predisposing factor for impairment of the following clinical dysfunction signs: subjects with overjets of 3.0 mm or less were more frequently associated with slight and severe mandibular mobility impairment, overjet of 3.5 mm or greater was associated with slight and severe TMJ function impairment, overjets of 3.5 mm or greater were associated with greater numbers of painful masticatory muscles.

In general, children and adolescents with overjets greater than 7 mm have been reported to also have associated joint sounds (Riolo, et al., 1987). All other studies found no relationship between overjet and the signs and symptoms of TMD (Lieberman et al., 1985; Shian, 1989; Gunn et al., 1987). In TMD patient populations, "large overjet appears to be a response to intracapsular arthrosis, and not an etiologic factor" (Seligman and Pullinger, 1991).

Even more so than overbite, the range of overjet found for completely asymptomatic subjects pretreatment was as wide as the whole sample. Almost 84% of these asymptomatic subjects pretreatment had a range of overjet from 0.5 to 4.0 mm. This agrees with Pullinger and Seligman (1991) whose results suggest that overjet because of its frequency and

wide range in nonpatient populations, is not a sensitive indicator of TMD.

Over 21% of the sample had some type of crossbite, which includes 6 (30%) subjects who were totally asymptomatic pretreatment. Crossbites in the present sample were associated with the following signs: slight mandibular mobility impairment, and slight TMJ function impairment which is consistent with autopsy findings which found greater deviation in TMJ form associated with crossbites (Solberg et al., 1986). Again, because of the small sample size and not being able to analyze the longitudinal data, it is difficult to suggest whether it was the crossbite which in fact caused the impairment or if it's correction would be of predictive value in terms of improvement of the associated sign.

Crossbites in young patients may predispose patients for future TMD depending on the adaptability of the patient, however, there is no basis for suspecting it as an etiologic factor in adults (Seligman and Pullinger, 1991). The transition from the mixed dentition to the adult dentition accounted for the high frequency of occlusal interference (Egermark-Eriksson et al., 1981; 1983), however, "occlusal interferences do not explain development or maintenance of TMD" (Egermark-Eriksson, et.al., 1987). Occlusal interferences in adults were approximately equally

distributed throughout all levels of dysfunction (Helkimo, 1974c).

Pullinger and others (1988b) found subluxation at maximum opening to be significantly associated with unilateral posterior crossbite, that is, 28% of the subjects with crossbite had this sign. Crossbite did not appear to be associated with TMJ pain.

Preponderance of the evidence available in terms of research supports the statement that in general, orthodontic treatment performed on children, adolescents, and adults does not put them at greater risk for the development of TMD in the future (Sadowsky, 1992). This conclusion fits well with the current philosophy of the etiology of TMD, that is, TMD is the result of a multitude of factors and can be exacerbated or alleviated by as many extrinsic factors. Secondly, orthodontic treatment is a slow process which does not exceed the adaptive capabilities of the human body. Finally, orthodontic treatment is a mechanotherapy process which in effect "splints" all the teeth together and progresses to improved occlusal and functional relationships.

Future of the Iowa TMD Study

The proposed study was to evaluate a longitudinal sample for risk factors: patient attributes, treatment modalities, and treatment results common to orthodontic

patients of 16 to 25 years of age. That point was never reached due to the small sample size in terms of similar pretreatment conditions, that is, similar pretreatment patient attributes, treatment modalities, and severity of pretreatment signs and symptoms. These criteria are necessary in order to distinguish statistically significant differences between the effects of patient attributes and treatment factors.

It became clear that the study was not in a position to close, but rather, it was just beginning to accumulate the necessary size in several important subgroups. Angle Class II patients treated with two upper bicuspid extraction was the largest group. It also became evident that there would not be sufficient numbers in some types of patients in my lifetime! Specifically, Angle Class III patients treated non-surgically. Approximately what size in terms of numbers would be needed to evaluate for risk factors? Janson and Hasund (1981) used 30 Angle Class II patients who were treated nonextraction and 30 Angle Class II patients treated with extractions, and compared them to an untreated control group, also of 30 subjects.

Knowing that this study has been ongoing for almost 10 years, that each year investigators aim to include 30 new patients, and that the largest group of subjects with similar pretreatment conditions and treatment modalities is

seven, then we can estimate that it may take 40 years to achieve the desired sample size!

What then are the alternatives? They include concentration on the specific case types in greatest numbers, statistical wizardry, or a new prospective study which would address the design problems in this study.

Is there any information this study can provide on risk factors? Possibly some important risk factors common to both pretreatment and posttreatment occlusions may be within grasp given that there are over 300 subjects in the study. That is, the evaluation of the entire sample across pretreatment patient attributes may identify some potential occlusal risk factors which are associated with pretreatment signs and symptoms.

There are other considerations which need to be evaluated in light of the small sample size. The most obvious is that the multifactorial nature of TMD makes it extremely difficult to determine specific etiologies. "The etiology of these problems is so complex that for a single patient, the specific cause(s) often can not be ascertained ... We need a new method to evaluate the problem" (Green (1984). There is a "need to move back to the concept of evaluation of the whole person" (Pullinger and Monteiro, 1988).

Unfortunately, at the beginning of this study, no data were collected with regard to the pretreatment functional occlusion. Clearly, our results indicate that functional determinants of occlusion, overbite, overjet, and crossbites do influence various components of the masticatory system, often having opposite effects.

The data also does not reflect in any way a history of habits such as bruxism, clenching, or other unusual biting habits. These are reported in the literature as possible etiologic factors to some forms of TMD.

Finally, the importance and role of stress factors and psychologic makeup or coping ability and their relationship to occlusal factors are unknown in the present study. On going research in this area has yet to identify psychological risk factors in TMD patients (Zach, 1992).

All three of these areas are filled with potential risk factors which have been excluded by the present method of evaluation.

In light of this information, it is quite probable that future information that the Iowa TMD Study may contribute may not justify the time, money and effort it entails. The best example of this scientific progress is the longitudinal study by Dibbetts and van der Weele (1987). It has been ongoing with modifications for over 20 years. Like the Iowa Study, parts of important information are missing which

compromises the interpretation of the information presented, and some of the information collected is no longer valid or current by today's standard of care, for example, panorex radiograph interpretations. It is in effect, obsolete!

Likewise, the Helkimo Indices were not designed for diagnosis, only epidemiologic descriptive survey studies. This study has used it as a means to evaluate changes in specific clinical dysfunction signs. This is an important consideration for the future of this study, especially in light of its lack of proven clinical validity.

It is clear that changes in occlusion have a more important role in some signs and symptoms of TMD than others. A majority of the patients remain unchanged from pretreatment to post treatment for each of the five signs investigated suggesting that occlusion has a minor role overall in the changes of TMD signs. At present, it is not known what treatment modalities could be avoided to prevent impairment, or what changes in patient attributes are beneficial.

CONCLUSION

From the results of this study, it can be concluded that :

1. The clinical dysfunction indices of the Helkimo Indices are a reliable method for measuring signs and symptoms in orthodontic patients. Examiners can remain consistent in examination technique, thereby maintain inter-examiner reliability.

2. The prevalence of pretreatment signs and symptoms of TMD as measured by the Helkimo Indices are within ranges reported in nonpatient populations, and previously studied postorthodontic treatment populations.

3. This study demonstrates the need for a large homogeneous sample and the importance of a longitudinal study to determine cause and effect relationships. In addition, there are other important etiologic factors of TMD, besides occlusal, which the Helkimo Indices do not address.

4. The signs of clinical dysfunction are affected to different degrees by orthodontic treatment.

- A. Impaired range of movement/mobility index

After one year into orthodontic treatment, 82.1% of the subjects improved or stayed at the same level of mobility impairment. After ending orthodontic treatment, 89.5% of the sample had improved or the same mandibular mobility.

B. Impaired TMJ function

After one year into orthodontic treatment, 87.3% of the subjects improved or stayed at the same level of TMJ impairment. After ending orthodontic treatment, however, 81.1% of the sample had improved or the same TMJ impairment.

C. Muscle pain

After one year into orthodontic treatment, 90.5% of the subjects had fewer or the same number of painful masticatory muscle sites. After ending orthodontic treatment, 88.4% of the sample had fewer or the same number of painful muscle sites.

D. TMJ Pain

After one year into orthodontic treatment, 90.6% of the subjects improved or stayed at the same level of TMJ pain. After ending orthodontic treatment, 93.7% of the sample had improved or the same TMJ impairment.

E. Pain on movement of the mandible

After one year into orthodontic treatment, 93.7% of the subjects had fewer or the same number of painful

mandibular movements. After ending orthodontic treatment, 94.7% of the sample had fewer or the same number of painful mandibular movements.

5. The severity of pretreatment signs of clinical dysfunction may be associated with the following pretreatment conditions: Angle Classification, crossbites, overbite, and overjet. However, none of these patient attributes appears to demonstrate the sensitivity or specificity which would enable their use as prediction factors for TMD or the impairment of the associated clinical signs.

APPENDIX A. THE HELKIMO INDICES FORMS

WRITTEN SUMMARY OF PATIENT PROCEDURES FOR IOWA
TEMPOROMANDIBULAR DISORDERS STUDY

You are being asked to participate in a research project which will assess the function of your mandible (lower jaw), and your temporomandibular joints (joints with the lower jaw to the remainder of the head). You will be given a brief questionnaire with questions regarding your lower jaw, teeth, and chewing muscles. After that, a brief examination will follow which will include inspection of your teeth, lower jaw movements, and the pair of TM joints. Your chewing muscles will be lightly palpated for any tenderness. The results of the collected data will be used in a research project to assess rates of occurrence of jaw movement problems in people of your age group, so that we can possibly improve dental treatment for you and others in the future.

All personal information will be held strictly confidential, and shall not be made public in any way. Your compliance in this project will not affect your treatment at the College of Dentistry. You may withdraw from the project at any time if you desire.

You will be informed of any appropriate treatment procedures which might be helpful to you if such a need is determined to exist. If you have any questions regarding the project, please contact Dr. John G. Kharouf at 351-7779 or at the Department of Orthodontics, 335-7308, College of Dentistry.

I have discussed the above points with the subject or his or her legally authorized representative, using a translator if necessary. It is my opinion that the subject understands the risk, benefits, and obligations involved in participation in this project.

Investigator

Date

Witness

IOWA TEMPOROMANDIBULAR DISORDERS STUDY
John G. Kharouf, D.D.S., Investigator
Department of Orthodontics
College of Dentistry
The University of Iowa

PATIENT CONSENT FORM

I, _____ have had a full explanation of the research project in which I or the person whom I represent as legal guardian will be participating.

I have been completely informed of the procedures to be used, along with any possible risks or benefits from the project, and have thoroughly read the attached written summary.

I understand that I have the right not to participate or withdraw at any time without loss of benefits to which I am entitled as a patient. I also understand that, if I am a student, my participation and/or withdrawal will not affect my grade in any course or my academic standing.

Signature

Date

Signature of legally authorized
representative if subject is under
18 years old

Date

I certify that I was present during the oral presentation of the attached summary when it was given to the subject or the legally authorized representative.

Witness

Date

Patient Personal History

We are requesting the following information so that we may contact you if necessary for follow-up treatment or research.

Patient number _____ Group _____

Name _____
Last First Middle initial

Age _____ Birth date _____ Sex _____ Race _____

Current Address _____
Number Street Apt #

City State Zip

Home phone () _____ Work phone () _____

Parents name _____
Last First Middle initial

Parents Address _____
Number Street Apt #

City State Zip

Debanded yes _____ no _____ Deband date _____
(For new Debands)

Initial visit _____ In treatment _____ Retention _____

Study _____

Comments

PATIENT HISTORY FORM FOR ANAMNESTIC INDEX

Patient number _____

Age _____

Group _____

Sex _____

1. Have you had previous orthodontic treatment? Yes___No___
If yes, how long ago was it? ___years ___months
2. Have you ever been treated for TMJ pain or dysfunction? Yes___No___
(Problems with your chewing muscles or jaw joint)
3. Do you often notice clicking or popping of your temporomandibular (jaw) joint? Yes___No___
4. Do your chewing muscles often feel tired or stiff when you awake Yes___No___
5. Do your chewing muscles often become sore or tender? Yes___No___
6. Do you have difficulty opening your mouth really wide? Yes___No___
7. Does your mouth ever lock open when you yawn or open it wide? Yes___No___
8. When you move your lower jaw from side to side, or open it wide, and close it does it cause pain? Yes___No___
9. Do you often experience pain in your jaw joints or chewing muscles? (Do not include toothache, headache, or pain of the neck or shoulder) Yes___No___
10. Have you ever experienced trauma or a blow to your lower jaw or jaw joints? Yes___No___

PATIENT HISTORY FORM FOR CLINICAL DYSFUNCTION INDEX

Angle classification _____ Patient number _____

Overbite _____ Overjet _____ Group _____

Mandibular mobility index

A. Maximal opening of mouth* _____ mm

≥ 40 mm 0

30-39 mm 1

< 30 mm 5

B. Maximal lateral movement to right _____ mm

≥ 7 mm 0

4-6 mm 1

0-3 mm 5

C. Maximal lateral movement to left _____ mm

≥ 7 mm 0

4-6 mm 1

0-3 mm 5

D. Maximal Protrusion _____ mm

≥ 7 mm 0

4-6 mm 1

0-3 mm 5

E. Sum A+B+C+D for mobility index = _____

F. Mobility index according to code _____

Code: 0 points: mobility index 0 or normal mandibular mobility
 1-4 points: mobility index 1 or slightly impaired mobility
 5-20 points: mobility index 5 or severely impaired mobility

*Maximal distance between edges of incisors + vertical overbite

Clinical dysfunction index, D_i , based on evaluation
of five different symptoms

<hr/>			
A. Symptom: Impaired range of movement/mobility index			
Criteria: Normal range of movement			0
Slight impaired mobility			1
Severely impaired mobility			5
<hr/>			
B. Symptom: Impaired TM-joint function			
Criteria: Smooth movement without TM-joint sounds and deviation on opening or closing movements $\leq 2\text{mm}$			0
TM-joint sounds in one or both joints and/or deviation $\geq 2\text{mm}$ on opening or closing movements			1
Locking and/or luxation of the TM-joint			5
<hr/>			
C. Symptom: Muscle pain			
Criteria: No tenderness to palpation in chewing muscles			0
Tenderness to palpation in 1-3 palpation sites			1
Tenderness to palpation in 4 or more palpation sites			5
<hr/>			
D. Symptom: Temporomandibular joint pain			
Criteria: No tenderness to palpation			0
Tenderness to palpation laterally			1
Tenderness to palpation posteriorly			5
<hr/>			
E. Symptom: Pain on movement of the mandible			
Criteria: No pain on movement			0
Pain on one movement			1
Pain on two or more movement			5
<hr/>			
F. Sum A+B+C+D+E for dysfunction score (0-25 points) _____			
<hr/>			
G. Dysfunction group 0-5 according to code _____			
<hr/>			
H. Clinical dysfunction index, D_i , according to code			
Code:	0 points: Dysfunction group 0	clinically symptom-free	D_iO
	1-4 points: Dysfunction group 1	mild dysfunction	D_iI
	5-9 points: Dysfunction group 2	moderate dysfunction	D_iII
	10-13 points: Dysfunction group 3}		
	15-17 points: Dysfunction group 4}	severe dysfunction	D_iIII
	20-25 points: Dysfunction group 5}		

MUSCLE PALPATIONS

	Note + if painful, - if not (Palpated bilaterally)	
	Left	Right
Masseter Muscle	_____	_____
Posterior temporalis muscle	_____	_____
Anterior temporalis	_____	_____
Lateral pterygoid muscle	_____	_____
Median pterygoid muscle (extraoral)	_____	_____
Temporalis insertion (coronoid)	_____	_____
No pain at any site		_____

APPENDIX B. PRETREATMENT DATA

PRETREATMENT HELKIMO

95 =NUMBER PATIENTS

1=YES & 0=NO IN ANAMNESTIC INDEX

1=MALE; 2=FEMALE

ID#	GRP#	AGE	SEX	ANAMNESTIC INDEX										ANGLE CLASS	ANGLE DIV	OVER BITE	OVER JET
				1	2	3	4	5	6	7	8	9	10				
1	1	23	2	0	0	0	0	0	0	0	0	0	0	2	1	8	8
7	1	17	2	0	0	1	0	0	0	0	0	0	0	2	1	6	4
8	1	21	2	0	0	1	0	0	1	1	0	0	0	1		0	6.5
10	1	25	1	0	0	0	0	0	0	0	0	0	0	3		1	-1
11	1	21	2	0	0	1	0	0	1	1	0	0	1	2		4	3.5
13	1	17	1	0	0	0	0	0	0	0	0	0	0	1		3	3
14	1	16	2	0	0	1	0	0	0	0	0	0	0	2		1	3.5
15	1	17	2	0	0	1	0	1	0	0	0	0	0	2	2	7	5
19	1	23	2	0	0	0	0	0	0	0	0	0	0	2	2	4	1.5
20	1	25	1	0	0	1	1	1	0	0	1	1	0	2	2	6	2.5
22	1	25	1	0	0	0	0	1	0	0	0	0	0	2	1	9	4.5
25	1	23	2	0	0	0	0	0	0	0	0	0	0	2		6	3
26	1	17	2	0	0	1	0	1	1	0	0	1	0	3		1	1
29	1	16	2	0	0	1	0	0	0	0	0	0	0	2		2.5	2
35	1	20	2	0	0	1	0	0	0	1	0	0	0	1		3.5	2
36	1	17	1	0	0	0	0	0	0	0	0	1	1	2	2	4	1.5
38	1	16	1	0	0	0	0	0	0	0	0	0	0	1		2.5	1
42	1	16	2	0	0	0	0	0	1	0	0	0	0	1		4	5
44	1	22	2	0	0	1	1	1	0	0	0	1	0	2	1	5	5
62	3	16	2	1	0	0	0	0	0	0	0	0	1	2	1	2.5	2
63	3	19	2	0	0	0	0	0	0	0	0	0	0	1		3	0.5
65	3	16	2	0	0	0	0	0	0	0	0	0	0	1		3	3
68	3	19	1	0	0	0	0	0	0	0	0	0	1	1		4	0.5
70	3	24	1	0	0	0	0	0	0	0	0	0	0	1		3.5	2
71	3	21	2	0	0	0	0	0	1	0	0	0	0	2		7	2.5
72	3	21	2	0	0	0	0	0	0	0	0	0	0	1		0.5	1
77	3	19	2	0	0	0	0	0	0	0	0	0	0	2		5	4.5
78	3	25	1	0	0	0	0	0	0	0	0	0	1	1		0	0
80	3	24	1	0	0	1	1	0	0	0	0	0	0	2		5	5
81	3	25	2	0	0	0	0	0	0	0	0	0	0	1		6	2
83	3	17	2	0	0	1	1	0	1	0	0	0	0	1		3.5	1
85	3	25	2	0	0	1	0	0	0	0	0	0	0	1		5	0.5
87	3	20	2	0	0	0	0	0	0	0	0	0	0	2		4.5	5
88	3	24	1	0	0	1	1	0	0	0	0	0	0	2	1	3.5	4
89	3	22	1	1	0	0	0	0	0	1	0	0	0	2	1	5.5	4
90	3	23	2	0	0	0	0	0	0	0	0	0	0	2		5	3.5
92	3	23	2	0	0	0	0	0	0	0	0	0	0	1		3.5	2.5
95	6	16	2	0	0	0	0	0	0	0	0	0	0	2		6	4
97	6	18	2	0	0	0	0	0	0	0	0	0	0	1		2	5
100	6	21	2	0	0	0	0	0	0	0	0	0	0	2		1.5	2
101	6	18	2	0	0	0	0	0	0	0	0	0	0	3		3.5	0.5
105	6	18	1	0	0	0	0	0	0	0	0	0	0	1		3	4
108	6	20	1	0	0	0	0	0	0	0	0	0	0	2		-2	3.5
109	6	18	2	0	0	0	0	0	0	0	0	0	0	1		1	2.5
110	6	19	1	0	0	0	0	0	0	0	0	0	1	1		2	1

113	6	16	2	0	0	0	0	0	0	0	0	0	0	0	2	2	4.5	1
114	6	17	1	0	0	1	0	0	0	0	0	0	0	1	1		4	3
115	6	16	2	0	0	0	0	0	0	0	0	0	0	0	1		2	2.5
116	6	23	2	0	0	0	0	0	0	0	0	0	0	0	2	1	4	7
118	6	16	2	0	0	1	0	1	1	0	0	1	0	0	2		3	0.5
119	6	16	1	0	0	0	0	0	0	0	0	0	0	0	2	1	4	4
123	10	16	2	0	0	1	1	1	1	0	1	1	1	1	2		0.5	6
126	10	16	2	0	0	0	0	0	0	0	0	0	0	0	2		4	2.5
128	10	16	2	0	0	0	0	0	1	1	0	0	0	0	2		6	3
131	10	26	2	0	0	0	0	0	0	0	0	0	0	0	2	2	6	0.5
132	10	16	2	0	0	1	0	0	1	1	0	0	0	0	1		3	3.5
136	10	16	2	0	0	0	0	0	0	0	0	0	0	0	2	2	3	2.5
137	10	16	2	0	0	0	0	0	0	0	0	0	0	0	2		3.5	3.5
141	10	17	2	0	0	0	0	0	0	0	0	0	0	0	2	2	3.5	1
143	10	19	1	0	0	1	0	0	0	0	0	0	0	0	3		3	0.5
145	10	15	1	0	0	0	0	0	0	0	0	0	0	0	2	1	5	10
147	10	22	2	0	0	1	0	0	1	1	0	0	1	1	2	1	3	2.5
148	10	17	1	0	0	0	0	0	0	0	0	0	0	1	2		4	0.5
149	10	15	1	0	0	0	0	0	0	0	0	0	0	1	2		5	3
150	10	15	2	0	0	0	0	0	0	0	0	0	0	0	2		0.5	4
151	10	25	2	0	0	1	0	1	0	0	0	1	1	1	2	1	5	3
152	10	15	1	0	0	0	0	0	0	0	0	0	0	0	1		4	1.5
153	10	17	2	0	0	0	0	0	1	0	0	0	0	0	1		2	0.5
156	10	15	2	0	0	0	0	0	0	0	0	0	0	0	2		4	6
161	15	20	1	1	0	0	0	0	0	0	0	0	0	1	2		0.5	4
163	15	16	2	1	0	0	0	0	0	0	0	0	0	0	2	1	4	5
173	15	15	2	0	0	1	0	0	0	0	0	0	0	0	2	2	2	3
177	15	21	1	0	1	1	0	0	0	0	0	0	0	0	1		4	7
182	15	20	2	0	0	0	0	0	0	0	0	0	0	0	1		3	1
185	15	17	2	0	0	0	1	0	0	0	0	0	0	0	1		2	0.5
186	15	25	2	0	0	1	1	1	1	0	1	1	0	0	3		0	0
188	15	16	1	0	0	0	0	1	0	0	0	0	0	1	2	2	6	1.5
189	15	24	2	1	0	0	0	0	0	0	0	0	0	0	2	1	5	2.5
190	15	25	2	0	0	0	0	0	0	0	0	0	0	0	1		3.5	0.5
193	15	15	2	0	0	0	0	0	0	0	0	0	0	0	1		1	4.5
194	15	16	1	0	0	0	0	0	0	0	0	0	0	1	1		-1	7
195	15	19	2	0	0	0	0	0	0	0	0	0	0	0	2	1	3	3
197	21	22	1	0	0	0	0	0	0	0	0	0	0	1	1		5	2.5
198	21	21	2	0	0	1	0	0	0	0	0	0	0	0	1		5	1
201	21	15	2	0	0	0	0	0	0	0	0	0	0	0	2	2	4	2
202	21	24	1	0	0	0	0	0	0	0	0	0	0	0	2	1	3	3.5
204	21	25	2	0	0	0	0	0	0	0	0	0	0	0	2	1	1	2
205	21	16	2	0	0	0	0	0	0	0	0	0	0	0	1		4	2
212	21	24	1	0	0	0	0	0	0	0	0	0	0	1	1		4	1
213	21	16	1	0	0	1	0	0	0	0	0	0	0	0	2	1	3	1
214	21	16	1	0	0	0	0	0	0	0	0	0	0	0	2	1	5	3.5
220	21	15	2	0	0	0	0	1	0	0	0	0	0	0	2	2	5	1
223	21	21	2	0	0	0	0	0	0	0	0	0	0	0	2	1	2	3
296	36	35	2	0	0	0	0	0	0	0	0	0	0	0	2	2	3	1
305	36	16	1	0	0	1	0	0	0	0	0	0	0	0	1		2	2

ID#	HELK DATE
1	7/26/83
7	8/22/83
8	8/23/83
10	8/26/83
11	8/31/83
13	9/2/83
14	9/6/83
15	9/8/83
19	9/14/83
20	9/15/83
22	9/19/83
25	9/21/83
26	9/28/83
29	10/4/83
35	10/13/83
36	10/18/83
38	10/19/83
42	10/27/83
44	11/9/83
62	7/18/84
63	7/18/84
65	7/20/84
68	7/23/84
70	7/24/84
71	7/25/84
72	7/25/84
77	7/27/84
78	7/27/84
80	7/31/84
81	8/1/84
83	8/2/84
85	8/3/84
87	8/3/84
88	10/2/84
89	10/5/84
90	10/25/84
92	12/5/84
95	9/17/85
97	7/12/85
100	9/17/85
101	9/3/85
105	8/9/85
108	9/27/85
109	9/10/85
110	9/24/85

113	7/16/85
114	9/11/85
115	9/19/85
116	7/25/85
118	10/24/85
119	7/18/85
123	7/23/86
126	8/1/86
128	7/16/86
131	7/25/86
132	9/23/86
136	7/24/86
137	7/25/86
141	8/5/86
143	7/31/86
145	7/29/86
147	9/28/86
148	7/24/86
149	9/24/86
150	7/17/86
151	7/17/86
152	7/24/86
153	8/5/86
156	7/31/86
161	8/24/87
163	7/30/87
173	7/23/87
177	7/24/87
182	7/23/87
185	7/24/87
186	7/29/87
188	7/22/87
189	9/28/87
190	7/28/87
193	7/21/87
194	7/21/87
195	7/23/87
197	9/2/88
198	8/29/88
201	8/3/88
202	9/14/88
204	9/9/88
205	8/3/88
212	9/2/88
213	8/4/88
214	10/31/88
220	8/10/88
223	9/1/88
296	9/7/90
305	10/11/90

APPENDIX C. DURING TREATMENT DATA

1st IN TREATMENT HELKIMO

N=95

1=YES & 0=NO IN ANAMNESTIC INDEX

1=MALE; 2=FEMALE

ID#	GRP#	AGE	SEX	ANAMNESTIC INDEX										ANGLE	OVER BITE	OVER JET
				1	2	3	4	5	6	7	8	9	10			
1	4	24	2	1	0	0	0	0	0	0	0	0	0	2	5	4
7	4	18	2	1	0	1	0	0	0	0	0	0	0	1	3.5	3.5
8	4	22	2	1	0	1	1	0	1	0	0	0	0	1	2.5	4
10	4	26	1	1	0	0	0	0	0	0	1	0	0	3	2.5	2
11	4	22	2	1	0	1	0	0	1	1	0	0	0	1	4	3
13	4	18	1	1	0	0	0	0	0	0	0	0	0	1	3.5	1.5
14	4	17	2	1	0	1	0	0	0	0	0	0	0	1	1.5	0.5
15	4	18	2	0	0	1	1	0	0	0	0	1	0	1	5	5.5
19	4	24	2	1	0	0	0	0	0	0	0	0	0	1	2	4
20	4	27	1	1	0	1	1	1	0	0	1	1	0	1	2	1.5
22	4	26	1	1	0	0	0	0	0	0	0	0	0	2	2	3
25	4	24	2	1	0	0	0	0	0	0	0	0	0	1	6.5	4
26	4	18	2	1	0	1	0	1	0	0	0	1	0	1	-1	1
29	4	17	2	1	0	0	0	0	0	0	0	0	0	1	2	1
35	4	21	2	1	0	0	0	0	0	0	0	0	0	1	1	1
36	4	18	1	1	0	0	0	0	0	0	0	0	1	1	0	0.5
38	4	17	1	1	0	1	0	1	0	0	0	0	0	3	-3	-2
42	4	16	2	1	0	0	0	1	1	0	0	1	0	1	4.5	4
44	4	23	2	1	0	1	0	0	0	0	0	0	1	1	4	5
62	7	17	2	0	0	0	0	0	0	0	0	0	0	1	4	5
63	7	20	2	0	0	0	0	0	0	0	0	0	0	1	0	0
65	7	17	2	0	0	0	0	0	0	0	0	0	0	1	1	0
68	7	20	1	0	0	0	0	0	0	0	0	0	0	1	1	1
70	7	25	1	0	0	0	0	0	0	0	0	0	0	1	2	1.5
71	7	22	2	0	0	0	0	0	0	0	0	0	0	1	1.5	1
72	7	22	2	0	0	0	0	0	0	0	0	0	0	1	0.5	0.5
77	7	20	2	0	0	0	0	0	0	1	0	0	0	2	4.5	6
78	7	26	1	1	0	0	0	0	0	0	0	0	1	1	2	1.5
80	7	25	1	0	0	1	0	0	0	0	0	0	0	1	0.5	2
81	7	26	2	0	0	0	0	0	0	0	0	0	0	2	1.5	6
83	7	18	2	1	0	1	1	1	1	0	1	0	0	1	2	2.5
85	7	26	2	1	0	0	0	0	0	0	0	0	0	1	2	1.5
87	7	21	2	0	0	0	1	0	1	0	0	0	0	2	1.5	4
88	7	24	1	0	0	0	1	1	1	0	0	0	0	1	2	3
89	7	22	1	1	0	0	0	0	0	0	0	0	0	1	3	3.5
90	7	24	2	0	0	0	0	0	0	0	0	0	0	1	4	4
92	7	24	2	0	0	0	0	0	0	0	0	0	0	1	2	2
95	11	17	2	0	0	0	0	0	0	0	0	0	0	1	2	4
97	11	20	2	0	0	0	0	0	0	0	0	0	0	1	2	4
100	11	21	2	0	0	0	0	0	0	0	0	0	0	1	-0.5	0
101	11	19	2	1	0	1	0	0	0	0	0	0	0	3	0.5	1.5
105	11	19	1	0	0	0	0	0	0	0	0	0	0	1	2	0.5
108	11	21	1	0	0	0	0	0	0	0	0	0	0	2	2	2
109	11	19	2	0	0	0	0	0	0	0	0	0	0	1	1	0.5
110	11	20	1	0	0	0	0	0	0	0	0	0	0	1	1.5	1

1=YES & 0=NO IN MUSCLE PALPATATIONS

CLINICAL DYSFUNCTION:

PHYSICAL EXAMINATION																							
MOBILITY					SYMPTOM					MUSCLE PALPATATIONS													
ID#	A	B	C	D	A	B	C	D	E	L1	L2	L3	L4	L5	L6	R1	R2	R3	R4	R5	R6		

1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0		
7	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0		
8	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0		
10	0	0	0	0	0	1	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0		
11	0	0	0	0	0	5	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0		
13	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0		
14	1	1	0	0	1	1	1	5	0	0	0	0	1	0	0	0	0	0	0	0	0		
15	0	0	1	0	1	1	1	5	1	1	0	0	0	0	0	0	0	0	0	0	0		
19	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	1	0		
20	0	0	0	0	0	1	1	5	1	1	0	0	0	0	0	0	0	0	0	0	0		
22	0	1	1	0	1	1	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0		
25	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0		
26	0	0	0	0	0	1	1	0	0	0	0	0	1	0	0	0	0	0	0	0	0		
29	0	0	0	0	0	1	1	0	0	0	0	0	0	1	0	0	0	0	0	0	1		
35	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0		
36	0	0	1	0	1	0	5	5	0	0	0	0	0	1	0	1	0	1	1	1	0		
38	0	0	0	0	0	0	1	5	0	0	0	0	0	0	0	0	0	1	0	1	0		
42	0	0	0	0	0	0	1	1	0	0	1	0	1	0	0	0	0	0	1	0	0		
44	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0		
62	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0		
63	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0		
65	0	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0		
68	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0		
70	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0		
71	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0		
72	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0		
77	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0		
78	0	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0		
80	0	0	0	1	1	1	0	0	0														

ID#	HELK DATE
1	11/16/84
7	10/10/84
8	10/16/84
10	9/28/84
11	10/17/84
13	11/16/84
14	10/15/84
15	9/27/84
19	10/10/84
20	11/1/84
22	10/23/84
25	10/10/84
26	10/23/84
29	11/1/84
35	10/18/84
36	11/7/84
38	10/26/84
42	9/27/84
44	10/10/84
62	9/11/85
63	9/5/85
65	7/26/85
68	8/5/85
70	7/16/85
71	8/6/85
72	7/17/85
77	7/24/85
78	7/26/85
80	8/8/85
81	7/17/85
83	9/19/85
85	7/31/85
87	7/22/85
88	7/16/85
89	7/24/85
90	7/18/85
92	7/31/85
95	8/26/86
97	8/26/86
100	7/18/86
101	7/22/86
105	7/23/86
108	7/25/86
109	9/2/86
110	9/2/86

113 8/5/86
114 7/31/86
115 8/6/86
116 7/17/86
118 7/10/86
119 7/24/86
123 8/27/87
126 7/16/87
128 9/4/87
131 7/8/87
132 8/20/87
136 8/12/87
137 9/17/87
141 7/14/87
143 7/6/87
145 7/14/87
147 7/17/87
148 7/21/87
149 7/9/87
150 7/11/87
151 7/9/87
152 8/27/87
153 7/30/87
156 7/17/87
161 9/8/88
163 8/31/88
173 9/15/88
177 9/9/88
182 9/23/88
185 9/8/88
186 9/6/88
188 12/8/88
189 10/11/88
190 9/20/88
193 10/5/88
194 9/14/88
195 9/20/88
195 8/29/89
198 8/22/89
201 8/22/89
202 8/25/89
204 9/28/89
205 8/22/89
212 8/25/89
213 10/5/89
214 10/27/89
220 8/25/89
223 9/28/89
296 7/16/91
305 7/31/91

APPENDIX D. POSTTREATMENT DATA

1st POST DEBAND HELKIMO

N=95

1=MALE; 2=FEMALE

ID#	GRP#	AGE	SEX	ANAMNESTIC INDEX										CLASS	ANGLE	OVER	OVER
				1	2	3	4	5	6	7	8	9	10				
1	13	26	2	1	0	0	0	0	0	0	0	0	0	1	2	1	
7	8	19	2	0	0	1	0	0	0	0	0	0	0	1	4	1.5	
8	13	24	2	1	0	0	0	0	1	0	0	0	0	1	2	1.5	
10	8	27	1	0	0	0	0	0	0	0	0	0	1	1	1	1.5	
11	8	23	2	1	0	0	0	0	1	1	0	0	0	1	1	2.5	
13	8	19	1	0	0	0	0	0	0	0	0	0	0	1	4	2	
14	8	18	2	1	0	1	0	0	0	0	0	0	0	2	2	1	
15	19	21	2	0	0	1	0	0	0	0	1	1	1	1	3.5	2	
19	8	25	2	0	0	0	0	0	0	0	0	0	0	1	1.5	1.5	
20	8	28	1	1	0	1	1	1	0	0	0	1	0	1	3	1.5	
22	8	27	1	1	0	0	0	0	0	0	0	0	0	1	3.5	1	
25	13	26	2	1	0	0	0	0	0	0	0	0	0	1	4	1	
26	8	20	2	1	0	1	0	0	1	0	0	0	0	1	1.5	1.5	
29	8	18	2	0	0	0	0	0	0	0	0	0	0	1	2	0.5	
35	8	22	2	1	0	0	0	0	0	0	0	0	0	1	3	1	
36	8	19	1	0	0	0	0	0	0	0	0	0	1	1	2.5	1	
38	13	19	1	0	0	0	0	0	0	0	0	0	0	3	-1	-1	
42	13	18	2	0	0	0	0	0	1	0	0	0	1	1	2.5	1.5	
44	13	25	2	1	0	0	1	1	0	0	0	1	1	1	4	1.5	
62	25	21	2	1	0	1	1	0	0	0	0	0	1	1	4	1	
63	18	22	2	1	0	0	0	0	0	0	0	0	0	1	2.5	1	
65	12	18	2	0	0	0	0	0	0	0	0	0	0	1	1.5	0.5	
68	12	21	1	0	0	0	0	0	0	0	0	0	0	1	2.5	0.5	
70	12	26	1	0	0	0	0	0	0	0	0	0	0	1	3	2.5	
71	12	22	2	1	0	0	0	0	0	0	0	0	0	1	3	2	
72	12	23	2	0	0	0	0	0	0	0	0	0	0	1	2.5	2	
77	18	22	2	0	0	0	0	0	0	0	0	0	0	1	3.5	1.5	
78	12	27	1	0	0	0	0	0	0	0	0	0	1	1	4	1	
80	25	29	1	0	0	1	0	0	0	0	0	0	0	2	3	2	
81	18	28	2	0	0	1	0	0	0	0	0	0	0	1	3.5	1.5	
83	18	20	2	0	0	0	1	1	0	0	0	0	0	1	2.5	1	
85	12	27	2	1	0	1	1	1	0	0	0	1	0	1	3	2.5	
87	18	23	2	0	0	0	0	0	0	0	0	0	1	1	3	1.5	
88	12	25	1	1	0	0	0	1	0	0	0	0	0	1	2.5	3	
89	12	23	1	1	0	0	0	0	0	0	0	0	0	1	1.5	1	
90	12	25	2	1	0	0	0	0	0	0	0	0	0	1	3	1.5	
92	12	25	2	1	0	0	0	0	0	0	0	0	0	1	2.5	1.5	
95	17	18	2	0	0	0	0	0	0	0	0	0	0	1	3	0.5	
97	17	21	2	0	0	0	0	0	0	0	0	0	0	1	2	3	
100	17	22	2	0	0	0	0	0	0	0	0	0	0	1	0.5	1	
101	17	20	2	0	0	1	0	0	0	1	0	0	0	1	5	0.5	
105	24	22	1	0	0	0	0	0	0	0	0	0	0	1	1	2	
108	32	25	1	0	0	0	0	0	0	0	0	0	0	1	1.5	1	
109	17	20	2	0	0	.	1	1	0	0	1	1	0	1	1.5	1	
110	24	23	1	1	0	0	0	0	0	0	0	0	0	1	1	4	

1=YES & 0=NO IN MUSCLE PALPATATIONS

CLINICAL DYSFUNCTION:

ID#	MOBILITY SYMPTOM					MUSCLE PALPATATIONS															
	A	B	C	D	E	L1	L2	L3	L4	L5	L6	R1	R2	R3	R4	R5	R6				
*****	*****	*****	*****	*****	*****	*****	*****	*****	*****	*****	*****	*****	*****	*****	*****	*****	*****	*****			
1	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
7	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
8	1	0	0	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	
10	0	0	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
11	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
13	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
14	0	1	1	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	
15	0	0	0	0	0	1	1	0	1	0	0	0	0	0	1	0	0	0	0	0	
19	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
20	0	0	0	0	0	1	1	0	1	0	1	1	0	1	0	0	0	0	0	0	
22	0	1	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
25	0	0	0	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
26	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
29	0	0	0	1	1	1	0	0	0	0	0	0	1	0	1	0	0	1	0	1	
35	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
36	0	0	1	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
38	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
42	1	0	0	0	1	0	5	0	0	0	0	0	1	1	1	0	0	0	1	0	
44	0	0	0	0	0	1	1	0	0	0	0	0	0	1	0	0	0	0	0	0	
62	0	0	0	0	0	1	1	1	0	0	0	0	0	1	0	0	0	0	0	0	
63	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
65	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
68	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
70	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
71	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
72	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
77	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
78	0	0	0	0	0	0	1	0	1	0	0	0	0	1	0	0	0	0	1	0	
80	0	1	0	0	1	1	0	0	1	0	0	0	0	0	0	0	0	0	0	0	
81	0	0	0	0																	

ID#	HELK DATE
1	7/3/86
7	12/4/85
8	7/16/86
10	9/25/85
11	9/4/85
13	1/7/86
14	10/1/85
15	7/17/87
19	5/28/86
20	3/6/86
22	9/17/85
25	6/2/86
26	12/20/85
29	9/19/85
35	11/4/85
36	3/11/86
38	9/2/86
42	10/15/86
44	9/15/86
62	12/5/88
63	10/12/87
65	6/23/86
68	8/5/86
70	8/27/86
71	6/2/86
72	7/27/86
77	9/16/87
78	9/2/86
80	9/16/88
81	7/24/87
83	9/2/87
85	7/22/86
87	7/17/87
88	7/28/86
89	9/19/86
90	2/21/87
92	8/4/86
95	7/21/87
97	10/30/87
100	10/19/87
101	10/29/87
105	11/16/88
108	1/2/90
109	7/15/87
110	1/27/89

113 7/17/87
114 11/4/87
115 8/27/87
116 9/3/87
118 9/10/87
119 9/24/87
123 9/27/88
126 12/23/88
128 4/24/89
131 8/30/88
132 8/31/88
136 3/1/90
137 12/23/88
141 11/1/88
143 7/12/89
145 10/5/88
147 8/17/89
148 9/7/88
149 11/10/89
150 10/28/88
151 9/9/88
152 11/10/88
153 9/22/88
156 9/2/88
161 11/30/90
163 11/6/89
173 7/13/89
177 3/29/89
182 5/23/89
185 4/14/89
186 1/19/90
188 11/2/90
189 9/14/89
190 11/14/89
193 1/7/90
194 3/13/90
195 1/5/90
197 10/31/90
198 10/26/90
201 9/25/91
202 10/18/90
204 7/11/90
205 8/27/90
212 1/16/90
213 6/25/91
214 8/30/91
220 7/13/90
223 3/1/90
296 1/14/92
305 12/16/91

APPENDIX E. POSTTREATMENT PERIOD II DATA

ID#	GRP#	AGE	SEX	ANAMNESTIC INDEX										ANGLE OVER OVER		
				1	2	3	4	5	6	7	8	9	10	CLASS	BITE	JET
1	19	27	2	0	0	0	0	0	0	0	0	0	0	1	3	1
7	13	20	2	0	0	0	0	0	0	0	0	0	0	1	3.5	0.5
8	19	25	2	0	0	1	0	0	1	0	0	0	0	1	2	1
10	13	27	1	1	0	0	0	0	0	0	0	0	1	1	2.5	1
11	13	24	2	1	0	0	0	0	1	1	0	0	0	1	2.5	2.5
13	13	20	1	1	0	0	0	0	0	0	0	0	0	1	3	1.5
14	13	19	2	1	0	1	0	0	0	0	0	0	0	1	3	0.5
15	26	22	2	1	0	0	0	0	0	0	0	0	0	1	3	1.5
19	13	25	2	0	0	0	0	0	0	0	0	0	0	1	3	1
20	13	29	1	1	0	1	1	1	0	0	1	1	0	1	3	1.5
22	13	28	1	1	0	0	0	0	0	0	0	0	0	1	3.5	2
25	19	27	2	0	0	0	0	0	0	0	0	0	0	1	5	1.5
26																
29	13	18	2	0	0	1	0	1	1	0	1	1	0	1	1.5	1
35	13	23	2	1	0	0	0	0	0	0	0	0	0	1	3	1
36																
38	34	22	1	1	0	0	0	0	0	0	0	0	0	3	0	-1.5
42	26	21	2	1	0	0	0	0	1	0	0	0	0	1	3.5	2
44	26	27	2	1	0	0	0	0	0	0	0	0	1	1	4.5	2
62																
63																
65																
68																
70																
71																
72	33	26	2	1	0	0	0	0	0	0	0	0	0	1	3.5	0.5
77	25	23	2	1	0	0	0	0	0	0	0	0	0	1	4	2.5
78	18	28	1	0	0	0	0	0	0	0	0	0	1	1	4	0.5
80	33	29	1	1	0	1	0	0	0	0	0	0	1	2	3	3
81																
83	25	21	2	0	0	1	1	0	0	0	0	0	0	1	3	1.5
85	18	28	2	0	1	1	1	1	0	0	0	1	0	1	4	1.5
87	25	25	2	1	0	0	0	0	0	0	0	0	0	1	5	2
88	18	27	1	0	0	0	0	0	1	0	0	0	0	1	2.5	2.5
89	18	24	1	1	0	0	0	0	0	0	0	0	0	1	2	1
90	18	26	2	0	0	0	0	0	0	0	0	0	0	1	3.5	1.5
92																
95	41	21	2	1	0	0	0	0	0	0	0	0	0	1	4.5	0
97	24	22	2													


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113 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0
114 0 0 0 0 0 1 1 1 0 0 0 0 1 0 0 0 0 1 0 0
115 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0
116 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0
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119 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0
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126 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0
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131 0 0 0 1 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0
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141 0 0 0 1 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0
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189 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0
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198 0 1 1 0 1 0 1 0 0 0 0 0 0 1 0 0 0 0 0 0
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223 1 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0
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305

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ID#	HELK DATE
1	9/10/87
7	7/9/86
8	9/10/87
10	6/30/86
11	6/18/86
13	7/1/86
14	8/5/86
15	11/23/88
19	9/18/86
20	8/7/86
22	5/22/86
25	10/8/87
26	
29	5/22/86
35	7/18/86
36	
38	3/2/90
42	12/27/88
44	12/13/88
62	
63	
65	
68	
70	
71	
72	9/14/89
77	12/15/88
78	7/24/87
80	8/15/89
81	
83	10/6/88
85	11/4/87
87	12/28/88
88	12/3/87
89	8/3/87
90	7/30/87
92	
95	8/22/90
97	12/14/88
100	
101	8/10/88
105	6/5/89
108	7/1/91
109	9/1/88
110	

113 1/5/90
114 9/21/88
115 1/28/90
116 5/10/89
118 12/13/88
119 9/30/88
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126 6/26/89
128
131 11/8/89
132
136
137
141 10/11/89
143 10/17/90
145 6/21/89
147 9/17/90
148 3/30/89
149 11/1/90
150
151 12/1/89
152 1/31/91
153 7/12/89
156 10/5/90
161
163
173 8/29/90
177 3/6/90
182
185 6/15/89
186 11/19/90
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189 10/4/91
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193 10/3/90
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223 1/22/91
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305

APPENDIX F. TREATMENT MODALITIES AND TREATMENT LENGTH

EXTRACTION1= 4 BICUSPID

1= YES

2= 2 UPPER BICUSPID

0= NO

3= OTHER EXTRACTIONS

ID#	DEBAND DATE	INITIAL DATE	MONTHS IN TX	EXTR	HG	CL II	CL III	CROSS BITES
1	2/14/86	7/26/83	31	3	1	1	0	0
7	7/3/85	7/13/83	24	1	1	1	0	0
8	1/6/86	8/23/83	28	1	1	1	0	0
10	5/3/85	7/15/83	22	0	0	0	1	1
11	5/1/85	7/20/83	21	1	1	1	0	0
13	5/3/85	7/27/83	21	1	1	1	0	0
14	7/23/85	7/25/83	24	2	0	0	1	1
15	2/27/87	7/14/83	44	3	1	1	0	0
19	9/18/85	7/26/83	26	1	0	1	0	0
20	4/30/85	7/22/83	21	2	1	1	1	0
22	2/26/85	7/26/83	19	3	0	1	1	1
25	11/4/85	7/13/83	28	3	1	0	0	0
26	12/19/84	7/20/83	19	0	0	0	0	1
29	6/7/85	7/15/83	23	3	1	1	0	1
35	11/15/84	7/21/83	16	1	1	0	0	0
36	1/29/85	7/14/83	19	0	1	1	0	0
38	11/15/85	1/5/83	35	3	0	0	0	1
42	10/3/85	8/2/83	26	1	1	0	0	0
44	11/20/85	11/9/83	25	1	0	1	0	0
62	2/25/87	7/18/84	31	1	0	1	0	0
63	12/11/86	7/18/84	28	0	0	1	0	1
65	2/21/86	7/20/84	19	0	0	1	1	0
68	1/13/86	7/23/84	18	0	0	0	0	0
70	11/6/85	7/24/84	15	3	0	0	0	0
71	12/11/85	7/25/84	16	0	1	1	0	0
72	7/2/86	7/25/84	23	1	1	1	0	0
77	11/10/86	7/27/84	27	2	1	1	0	0
78	5/2/86	7/24/84	21	1	0	1	0	0
80	4/8/88	7/31/84	44	3	1	1	0	0
81	12/4/86	8/1/84	28	1	1	1	0	0
83	9/4/86	8/2/84	25	3	1	1	0	1
85	10/30/85	8/3/84	15	0	0	0	0	0
87	5/5/87	8/3/84	34	2	1	1	0	0
88	9/17/85	8/20/84	13	0	1	1	0	0
89	6/3/86	9/14/84	21	2	1	1	0	0
90	9/15/86	8/1/84	26	0	1	1	0	0
92	8/27/85	8/22/84	12	0	0	0	0	0
95	4/7/87	7/23/85	20	2	1	1	0	0
97	4/17/87	7/26/85	21	1	1	1	0	0
100	12/12/86	8/9/85	16	3	0	1	0	0
101	11/11/86	7/23/85	15	1	0	0	1	1
105	6/3/88	8/9/85	34	1	0	1	0	0
108	11/29/89	9/13/85	51	3	0	1	0	0
109	10/30/86	9/10/85	15	0	1	0	0	0
110	1/4/88	4/22/85	32	1	1	1	0	0

113	5/6/87	7/16/85	21	3	0	1	0	0
114	10/17/86	4/2/85	19	3	0	1	0	1
115	12/3/86	4/23/85	19	3	0	1	0	1
116	5/15/87	7/25/85	22	2	1	1	0	0
118	7/8/87	7/26/85	22	1	0	1	0	0
119	9/10/86	7/18/85	13	0	1	1	0	0
123	9/27/88	7/23/86	26	0	1	0	0	0
126	5/11/88	8/1/86	21	2	1	0	0	1
128	1/10/89	7/16/86	29	2	1	1	0	0
131	4/1/88	7/25/86	20	3	1	1	0	1
132	9/30/87	9/23/86	12	0	1	1	0	0
136	5/12/89	7/24/86	33	3	1	1	0	0
137	11/17/87	7/28/86	16	0	0	1	0	0
141	3/15/88	8/5/86	20	3	0	1	0	0
143	1/23/89	7/31/86	30	1	0	0	1	0
145	2/9/88	7/30/86	18	2	1	1	0	0
147	3/6/89	7/25/86	31	3	1	1	0	0
148	8/23/88	7/24/86	25	3	0	1	0	1
149	4/21/89	7/18/86	33	1	0	1	0	0
150	11/20/87	7/17/86	16	2	1	1	0	1
151	9/17/87	7/17/86	14	3	0	1	0	0
152	12/3/87	7/24/86	16	0	0	1	0	0
153	1/15/88	8/5/86	18	0	0	1	0	1
156	5/11/88	7/31/86	21	2	1	1	0	0
161	2/1/90	8/24/87	29	0	1	1	0	0
163	3/29/89	7/30/87	20	2	0	1	0	0
173	2/23/89	7/23/87	19	2	1	1	0	0
177	1/12/89	7/24/87	17	3	0	0	0	0
182	5/5/89	7/23/87	21	3	0	1	0	0
185	10/28/88	7/24/87	15	0	1	0	0	1
186	6/30/89	7/29/87	23	1	0	1	0	0
188	7/13/90	7/22/87	34	2	0	1	0	0
189	3/17/89	9/28/87	19	3	0	1	0	0
190	10/25/88	7/28/87	15	0	0	0	0	0
193	7/26/89	7/21/87	24	1	1	1	0	0
194	3/13/90	7/21/87	31	1	1	1	0	0
195	6/21/89	7/23/87	23	2	1	1	0	0
197	4/6/90	9/2/88	20	3	0	0	0	0
198	8/24/90	8/29/88	24	3	0	1	0	0
201	5/29/91	8/23/88	34	0	0	1	0	1
202	5/10/90	7/15/88	21	1	1	0	0	0
204	7/8/90	7/19/88	22	3	1	0	0	1
205	3/16/90	8/3/88	20	1	0	1	1	0
212	9/14/89	7/15/88	13	3	0	0	0	0
213	6/25/91	8/4/88	33	3	0	1	1	0
214	4/22/91	7/28/88	33	3	0	0	0	1
220	5/10/90	8/10/88	21	3	0	1	0	0
223	1/25/90	7/21/88	18	3	1	1	0	0
296	12/19/91	1/27/90	23	3	0	0	0	0
305	10/11/91	8/21/90	15	0	0	0	1	0

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